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of the

Forty-Third Annual Meeting

of the

American Academy of Ophthalmology and Otolaryngology

WASHINGTON, D. C.

October 9 to 14, 1938

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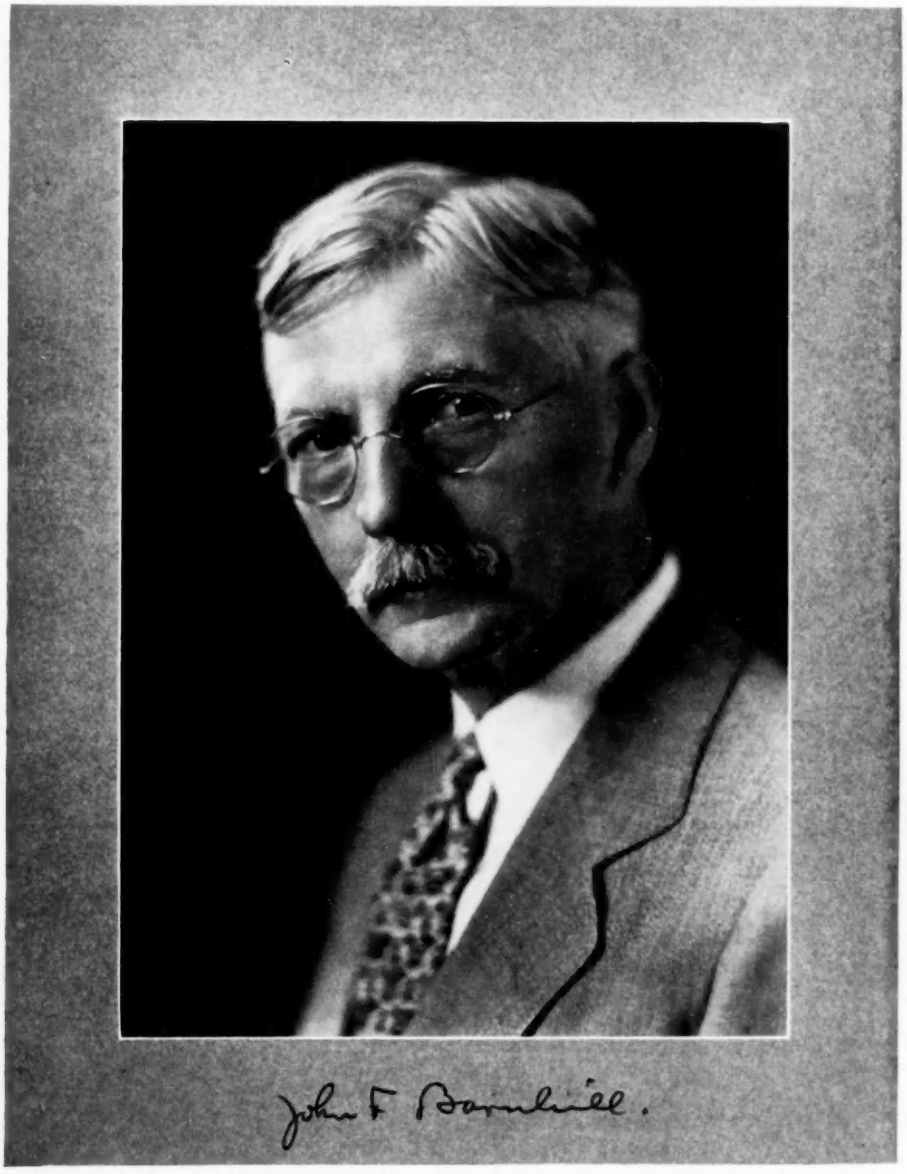

John F. Bamhill.

This Volume of the
Transactions of the American Academy of
Ophthalmology and Otolaryngology
Is Gratefully Dedicated to

John Finch Barnhill, M.D., F.A.C.S., LL.D.

President of the American Academy of Ophthalmology and Otolaryngology, 1932

INDIANAPOLIS

(Miami Beach, Florida, Since Retirement)

Doctor Barnhill was born in Illinois in 1865; he was educated in Indiana and graduated in medicine from Central College of Physicians and Surgeons. After an internship at Indianapolis Public Hospital he practiced general medicine and surgery in Indianapolis from 1890 to 1895. He took post-graduate work in otolaryngology in New York, London, Berlin and Vienna. Since then he has practiced otolaryngology and surgery of head and neck in Indianapolis.

He was Professor of Physiology at Central College of Physicians and Surgeons, 1891-1896; Professor of Otolaryngology, 1896-1907, Professor of Otolaryngology in Indiana University School of Medicine, 1907-1923; Professor of Surgery of the Head and Neck, Indiana University, 1923-1932, and Head of the Department of Otolaryngology. He conducted post-graduate courses in Cadaver Surgery of the Head and Neck, 1912-1939. He has held offices in many societies: Chairman, Section on Otolaryngology, A.M.A., 1904; President, Indiana Academy of Ophthalmology and Otolaryngology, 1923; President, Indianapolis Medical Society, 1924; President, American Laryngological, Rhinological and Otological Society, 1928; President, American Academy of Ophthalmology and Otolaryngology, 1932; President, American Laryngological Association, 1938, and Member of the American Otolaryngological Society.

Doctor Barnhill has written many articles, chiefly on surgical subjects. He is the author of textbooks on otolaryngology and several very interesting non-medical books, and is also Associate Editor of the Archives of Otolaryngology.

Doctor Barnhill has watched with increasing interest and tremendous pride the notable accomplishments of his confreres and he looks forward with confidence to a time when their continued efforts will have eliminated or at least greatly ameliorated much human suffering.

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Joint Session

The American Academy
of Ophthalmology and Otolaryngology

Forty-Third Annual Meeting
Washington 1938

Address of the President

* * *

POST-GRADUATE INSTRUCTION

HARRY S. GRADLE, M.D.
CHICAGO

PRESIDENCY of the American Academy of Ophthalmology and Otolaryngology implies an obligation for service far more than the conferring of a personal honor. While much of the actual labor connected with the office is adequately performed by the Board of Secretaries, to whom as a retiring member of eighteen years standing, I express my homage and devotion, there are other duties and responsibilities which I am happy to assume and to which I pledge my sincerest devotion.

The Academy has now grown beyond the stage of being a mere forum for the exchange of clinical experiences, the change in objectives dating from 1920 when the Section on Instruction was formed. At that time, the members instructed the officers of the Academy to provide means for post-graduate instruction, as a result of which the question was put into the hands of a Committee consisting of Dr. Hanau Loeb, Dr. Meyer Wiener, Dr. W. P. Wherry, and myself as Chairman. So the 1921 meeting in Philadelphia celebrated the birth of the Section on Instruction. The program, in the form of forensic lectures, was held in the large hall. Those lectures were attended by 650 members. The response was most gratifying and the Section expanded, increasing and amplifying the talks. The feature of Round Table discussions was introduced in 1925 and met with immediate favor.

This type of forensic instruction, however, was bound to be short-lived and in 1927, the program of the Section was changed so that the major portion of the instruction was offered in Conference Courses which have gradually developed to the present form. The work entailed in the Section on Instruction is enormous

and by experience it was found advisable to concentrate it in the office of two permanent Secretaries who could function more efficiently than a larger group. The two Secretaries prepare a preliminary program that is submitted to the Council at the annual program meeting when the entire Academy program is assembled and rounded into a tentatively acceptable form. Then the two Secretaries of Instruction, aided by the entire Secretarial Group, correlate the many details necessary for adequate presentation of the Instructional Courses.

At present there are 2,570 members of the Academy in the United States and Canada and 19 members representing 11 foreign countries. According to their own listings in the Academy Directory, 29 per cent (742 in number) devote their attention either exclusively or for the major part to Ophthalmology; 42 per cent (1,106 in number) to Otolaryngology; and 29 per cent (722 in number) practice both specialties. Of even greater interest is the geographical distribution of the Academy membership. Twenty-nine per cent of the membership is located in 480 communities of less than 50,000 population; only 9 per cent in some 52 communities of 50,000 to 100,000; 26 per cent in 54 communities of 100,000 to 500,000; and 36 per cent in some 13 communities of more than 500,000 population. Such a diversity of membership is a testimony to the efficiency with which the requirements of the members are being met.

What are the things that the members demand of their Society, the Academy? They demand that the Academy provide ways and means for the dissemination of knowledge in:

1. An open forum for the presentation and discussion of formal papers;
2. Panel discussions of topics of current professional interest;
3. Presentation of pictures, both still and moving, showing modern aspects of the specialties that lend themselves to this newer method of graphic demonstration;
4. Post-graduate instruction in the form of lectures and demonstrations;
5. Publications; the Transactions which contain the scientific contributions and the business activities of the Academy; and a Quarterly Bulletin with its multiple items of current interest.

The demand for post-graduate instruction is one which I wish to discuss at somewhat greater length. I have sketched briefly for

you the development of the Section on Instruction, which during the past eighteen years has presented instructional courses that have met with much favor. It is my wish that the instructional program be continued in an even more elaborate form than heretofore. The Academy can arrange to carry on opportunities for instruction so that the members may obtain the maximum in the time available. A present objection that the instruction is now more or less sporadic, can be overcome by a correlating agency through which instruction in the fundamental subjects will become more systematized and more thorough.

The fact must be recognized that not all members have had the same degree of preparation nor have they the same degree of comprehensive knowledge of the various aspects of our specialties. Consequently, instruction must be graduated to meet the various requirements. The Section has endeavored to do this in an informal way for the past seven or eight years, but the time is now ripe for the declaration of a graduated curriculum. Elementary courses in the fundamental subjects must be given for those who wish to review half-remembered knowledge. For a second group, there must be offered intermediate courses in basic and in clinical subjects. And finally we come to the new departure of advanced courses that should comprise the last word upon the subject in question. This matter has been discussed for many years by the Secretarial Board and now I beg leave to offer a plan for the further advancement of the Academy in post-graduate teaching. Accepting the idea of a graduated curriculum, I suggest that the Section on Instruction carry on as at present with the addition of a coordinating member who will assist the present Secretaries in planning the program for two years or more in advance, instead of only one year as is now done. Systematically graduated courses, for which members may register six months or more in advance of the meeting, will then constitute a part of each annual program. Contact between the Instructors and the Registrants will be established via the coordinator, resulting in directed home study of bibliographical references and microscopic preparations. Then in the courses presented at the annual meeting the instructors will not have to devote time to the introductory aspects or outlining the subject, for the fundamentals will have been covered in the directed home study preceding the course. The character of our post-graduate instruction will immediately step up beyond the plane of present post-graduate teaching.

There appear to be two logical further developments from this plan. In the first place, there should be set up within the Academy, a Faculty of Instruction, a form of recognition for those who have given courses and an inducement for others to teach. The list of such a faculty would be of enormous value to administrators of both graduate and post-graduate teaching. In the second place it would fall well within the function of the coordinating member of the section to act as a correlating agent between the teaching activities of the Academy, the various post-graduate courses that are being given throughout the country, the American Boards of Ophthalmology and Otolaryngology, and the institutions where graduate teaching is being conducted.

To one who has been actively engaged in serving as an Officer of this Academy, the conclusion of such service brings deep regrets. The work is one of love and pride in the Academy. But in order that this most virile organization may continue to progress for the benefit of its members and the greater good of mankind, new blood and new ideas are constantly required. So with deep thankfulness in my heart for what the Academy has done for me and with intense gratitude for the friendships that found their origin in the Academy, I paraphrase the words of the old Romans in a "*Vivat et floreat Academia.*"

Symposium on Dermatological Lesions
About the Eyes, Ears, Nose and Throat

* * *

DERMATOLOGICAL LESIONS ABOUT THE EYE

ROBERT VON DER HEYDT, M.D.
CHICAGO

A PERUSAL of ophthalmic textbooks, handbooks and literature on the subject of skin diseases involving the eye discloses a diversity in classification and nomenclature. This is but natural in view of the range of the field influenced by the personal clinical opportunities of the observers and their evaluation of the cases.

Many skin lesions invade the eyelids and adjacent areas and manifest themselves in much the same manner as on other parts of the body.

Of interest especially to ophthalmologists are the skin diseases in which the ocular involvement is peculiarly modified by the local anatomic and physiologic variations found in the lid margins, glands, conjunctiva and corneal epithelium. It is important to be able to recognize these often characteristic manifestations.

My presentation will consist of Kodachrome photographs of clinical cases made available only in the past seven months through the kind cooperation of local ophthalmologists and dermatologists. The series consequently may omit some of the skin lesions often included in a more complete list.

1. *Dermoid* at fornix.
2. *Dermoid* at corneal limbus.
3. *Cutaneous tags.*
4. *Marginal sebaceous cyst.*
5. *Verruca* or wart.

6. *Verruca* over sebaceous cyst.

7. *Xanthelasma.* Common benign symmetrical lid tumor. May be part of a general xanthomatosis in which the cholesterin content of blood may be increased.

8. *Atheroma.*

9. *Eczematous blepharitis.* (Eczema palpebrarum.)

10. *Contact dermatitis.* (Dermatitis venenata), due to external irritation from heat, light, medical, chemical or toxic agents.

11. *Contact dermatitis.* Another similar case.

12. *Contact dermatitis.* Quinine bisulphate in treatment of trachoma, also commonly seen in ophthalmological practice as an idiocyncrasy to long continued local use of atropine or eserine.

13. *Argyrosis* in a case of old trachoma.

14. *Ringworm.* Lid margin involvement by ringworm fungus — in a case of general involvement on the body.

15. *Ringworm.* Same case.

16. *Ringworm.* Same case upper lid surface lesions. All of these three lesions are specific ringworm infections.

17. *Fungus lesions* in colored boy, "Ide" eruption. A secondary local manifestation due to fungus lesions elsewhere on the body.

18. *Mulluscum contageosum.* These umbilicated lesions if close to the lid margin cause irritation of the conjunctiva.

19. *Ophthalmic herpes.* Acute involvement with lid edema.

20. *Ophthalmic herpes.* Lesions above eyebrow.

21. *Psoriasis of the scalp.* Hair line margin.

Four years ago Pillat published an article pertaining to rare cases of bilateral corneal involvement in psoriasis. The lesions were sector formed vascularized phlyctenular like efflorescenses at the limbus. These corneal lesions became acutely inflamed simultaneously with reactivation, of the general skin manifestations, hence Pillat is sure that they are related.

22. *Psoriasis.* Coincidental corneal lesion.

Dermatologists do not seem to recognize an ocular psoriasis. The psoriasis case shown in the color photograph was complicated by bi-

lateral non-inflammatory general clouding of the cornea at the Bowman level — not unlike band shaped keratitis. It seems doubtful if this peculiar corneal condition is related to psoriasis.

23. *Rosacea Keratitis.* Acute corneal limbus involvement of acne rosacea.

In addition the slitlamp disclosed snow flake spots scattered over the corneal epithelium. A most characteristic, often permanent, lesion is the development of large vascularized pinkish grey superficial plaques extending inward from the limbus. A papular acne of the face of the rosacea type may be present as well as seborrheic blepharitis.

24. *Acne Rosacea.* The patient's nose.

Characteristic corneal rosacea keratitis lesions may be found in individuals without nasal or cheek rosacea.

25. *Melanosis.* Mild intermittently hyperemic area adjacent to the limbus.

26. *Melanosis.* Blanching of blood vessels after instillation of adrenalin solution disclosing the pigment.

27. *Melanosis* of apex of lid margin granulation tissue.

28. *Melanosis* at limbus. (Pigmented naevus.)

29. *Melanosis* at semilunar fold. (Pigmented naevus) in colored woman.

30. *Pigmented naevus* near semilunar fold. Two photographs taken with same magnification, depicting increase in size of pigment area during a period of eleven months.

A naevus of this type is not a precancerous lesion. Acquired melanosis may become malignant.

31. *Melanosis.* A case of probable metamorphosis into malignancy.

In melanosis of the conjunctiva the onset of malignancy histologically is characterized by proliferation of the basal layer with possible hemorrhage and ulceration. Photographic records of the extent of melanotic involvements have a definite clinical importance. They may depict increases in size as well as the effects of therapy.

32. *Cataracta neurodermatica.*

A consideration of skin lesions of the eye would not be complete without mention of neurodermatogenous cataract. This type of cataract is usually seen in young persons in conjunction with atopic eczema. It is believed that endocrine gland disturbances or a disease of the sympathetic nerve may indirectly cause both the dermatosis and the lens change. The latter is characterized by an anterior subcapsular

often shield shaped central involvement. Its location proves its histologic dependence on disturbances of the lens epithelium.

The photograph shown is of the least involved eye. This young woman has had a brawny dermatitis (ichthyosis) since childhood. Thorough physical examination involving repeated metabolism tests failed to show other contributory causes. Both eyes were the subject of spontaneous hemorrhage into the anterior chamber. This eye at one time showed blood in the vitreous by slitlamp examination.

33. *Malignant lesions.*

34. *Epithelioma* (rodent ulcer).

35. *Epithelioma* (rodent ulcer).

36. *Epithelioma* (rodent ulcer).

37. *Epithelioma* at the limbus.

Symposium on Dermatological Lesions
About the Eyes, Ears, Nose and Throat

* * *

DERMATOLOGIC CONDITIONS OF THE EAR, NOSE AND THROAT

FREDERICK A. FIGI, M.D.
ROCHESTER, MINNESOTA

DERMATOLOGIC conditions of the ear, nose and throat may occur entirely as a local process; more commonly, however, they are a manifestation of some systemic disease. The large number of such conditions encountered makes it impossible to discuss all of them in a single paper, and only those of greatest clinical importance or of particular interest for other reasons will be considered here. The neoplasms, the acute exanthemas and some of the rare dermatologic conditions which only infrequently produce manifestations in these regions must of necessity be omitted.

Various systemic diseases and dermatoses are frequently accompanied by lesions of the ears, nose, mouth and throat. Glossitis and fissuring at the angles of the mouth often develop in secondary anemia, and a more or less characteristic atrophic glossitis is seen in pernicious anemia. Sensitive superficial erosions occur about the mouth and tongue in sprue. In the early stages of typhoid fever, submucous hemorrhages are commonly present in the nose and throat while, in the later stages, deep ulcerations may develop in these situations. Victims of pellagra and other vitamin deficiency diseases often have stomatitis and glossitis. Gingivitis and stomatitis (catarrhal) are often present in diabetes. Petechiae, and at times ecchymoses, may appear on the palate in purpura. Uremia may be accompanied by either an exudative or an ulcerative stomatitis.

While many acute infectious conditions occurring about the alveolar processes present a problem that is more distinctly dental

than medical, nevertheless the otolaryngologist should be thoroughly familiar with them. The lesions of Vincent's symbiosis, while almost invariably originating about the gingival borders, commonly involve other portions of the oral cavity and not infrequently the pharynx as well. A single, or multiple large ulcers on the base of the tongue may, in fact, be the most prominent manifestation of this condition.

The striking resemblance of the clinical picture in Vincent's infection to that at times seen in certain blood dyscrasias, especially acute and chronic lymphatic leukemia, often causes considerable difficulty from the standpoint of differential diagnosis. These conditions may be indistinguishable on the basis of the clinical examination alone, and a definite diagnosis should always be withheld in cases of this type until hemocytologic studies have been made even though smears from the gingival ulcerations reveal numerous Vincent's organisms.

In leukemia, especially of the lymphatic type, there may be, in addition to diffuse infiltration and ulceration of the gingiva, hypertrophy of the faucial and lingual tonsils, nodular infiltration of the palate or pharynx, or hemorrhagic, bullous, or necrotic lesions in these situations.

Agranulocytosis should also be mentioned in this connection. This disease, of undetermined etiology, is characterized by marked anemia, leukopenia and disappearance of the polymorphonuclear leukocytes from the blood. An increasing number of cases of this condition have been observed in recent years and the indiscriminate administration of certain coal tar products, especially amidopyrine, appears to have had some etiologic bearing. The usual lesion in agranulocytosis is a large sloughing ulcer, situated in the palate or pharynx, which tends to progress. While temporary improvement often occurs, remissions are frequent and the disease is commonly fatal.

The Plummer-Vinson syndrome (simple achlorhydric anemia) should be mentioned in this connection. This condition, which is on a nutritional basis, being due to iron deficiency, usually occurs in women. It is quite common in certain parts of Europe, especially in the Scandinavian countries among the poorer classes. It is characterized by dysphagia, secondary anemia, contraction of the mouth (with atrophy of the lips and fissuring at the angles), stomatitis, atrophic glossitis, atrophy and paleness of the pharyngeal and esophageal mucosa, and flat, brittle, spoon-shaped nails.

The mucous membranes of the mouth, pharynx, larynx and esophagus in these cases are very prone to undergo malignant change without previous leukoplakial thickening and multiple carcinomas are not infrequently seen in the mouth, pharynx and postcricoid region.

The infectious granulomas, syphilis, tuberculosis, actinomycosis, blastomycosis, rhinoscleroma, leprosy and yaws frequently produce pathologic changes in the upper respiratory and alimentary tracts. The lesions of primary, secondary and tertiary syphilis may appear about the nose, mouth, pharynx and ears. The primary sore, or chancre, is commonly seen on the borders of the lips; it may also occur on the nose, tongue, or elsewhere inside the mouth and it has been reported on the epiglottis. It is characterized by marked induration and foul ulceration, and the regional lymphatics are enlarged and tender. The most common manifestation of secondary syphilis in the mouth and throat is the mucous patch, although rashes similar to those occurring on the surface of the body, glossitis and persistent ulceropapular sore throat may occur. Tertiary syphilitic lesions in this region include gummas in various stages, scarring of the palate, pharynx or nasal mucosa, perforation of the palate or nasal septum, or collapse of the nose secondary to gummas, leukoplakia and chronic interstitial glossitis. Tertiary syphilis is a prominent factor in cicatricial stenosis of the nasopharynx. In the larynx, tertiary syphilis may present as gumma, ulceration, perichondritis, scarring or stenosis.

Tuberculosis may occur about the ear, nose and throat, either in its attenuated form, lupus, or as a true acute or chronic tuberculous lesion. Involvement of the nose by Mycobacterium tuberculosis is encountered almost exclusively as lupus; in the pharynx and ear, the process may exist either as lupus or as true tuberculosis; in the larynx, chronic tuberculous ulcers most often occur. The author has encountered involvement of the nasopharynx with a combination of tuberculosis and active squamous-cell epithelioma; the two conditions are at times coexistent in the larynx. In the ear, the infection most commonly produces a chronic suppurative otitis media.

Lupus occurs relatively infrequently in the United States as compared to the British Isles and continental European countries. About the face, the process commonly involves the nose, upper lip and lobes of the ears and often there is extension to the nasal and oral mucous membrane. The characteristic pinkish,

so-called apple-jelly nodules which usually first develop on the anterior portion of the septum often give way to ulceration and perforation of this structure. The process may heal in some areas while progressing in others.

Tuberculous lesions of the mouth and pharynx occur infrequently considering the prevalence of tuberculosis. They are most commonly found among individuals with advanced pulmonary involvement and usually appear as painful, superficial, moth-eaten ulcers which are usually multiple. They may involve any portion of the oral or pharyngeal mucous membrane and are likely to produce very little evidence of inflammatory reaction and induration in the surrounding tissues. In the mouth and pharynx, as well as in the larynx, these ulcers indicate a highly virulent infection, or lowered resistance to the infection on the part of the patient, and, as a rule, justify an unfavorable prognosis. About the jaws, a single, deep, indolent tuberculous ulcer with involvement of the bone at times occurs. Direct scrapings from the oral and pharyngeal ulcers will occasionally permit of demonstrating tubercle bacilli and thus of establishing a positive diagnosis.

Solitary tuberculoma may be encountered as a discrete tumor in the body of the tongue, lips, or cheeks, developing insidiously and painlessly. These present the characteristic histologic picture of tuberculosis, but their exact relation to pulmonary tuberculosis is indefinite. Roentgenograms of the thorax in cases of this type observed personally showed evidence of pulmonary involvement, but there was no sign of activity either clinically or roentgenographically. The histologic picture of gumma is practically indistinguishable from that of tuberculoma; in all of these cases, however, the serologic findings were negative. Excision was at times followed by recurrence, whereas irradiation was quite effective in controlling the condition.

Rhinoscleroma, or more properly scleroma, is a chronic infectious granuloma caused by the encapsulated Bacillus rhinoscleromatis or Frisch's bacillus. It occurs endemically among the inhabitants of central and southeastern Europe and sporadically in various other parts of the world, although chiefly among emigrants from these regions. A case of this type in which a native born American was affected was reported by Thompson and me in 1928. The condition is more common among adult females and is intimately associated with a lack of hygienic surroundings. The infection usually starts in the mucous mem-

brane of the anterior nares, the lesions appearing as edematous, bluish-red, discrete nodules which may remain localized in this region or spread onto the upper lip, the external nose, or into the palate, pharynx, larynx, or trachea. A large, ulcerated, granulomatous mass often protrudes from the nostrils and emits a foul fetid discharge so that the entire picture may strongly suggest a highly malignant neoplasm. The primary infiltration and hyperplasia is later followed by a sclerosing process and the mucous membrane becomes scarred and atrophic. A characteristic concentric stenosis of the nasopharynx, with retraction or destruction of the uvula, is commonly present and in itself is highly suggestive of scleroma. Marked deformity of the external portion of the nose and distortion of the larynx may also occur. The disease runs an extremely chronic course; it may continue for from fifteen to thirty years and then gradually subside spontaneously. The histopathologic picture, as emphasized by Montgomery, is diagnostic and consists of: (1) the presence of Mikulicz or foam cells containing Frisch's encapsulated bacillus; (2) numerous plasma cells, and (3) so-called Russell bodies. Irradiation is the most effective form of therapy. Culture of a biopsy specimen with demonstration of Klebsiella rhinoscleromatis clinches the diagnosis.

Actinomycosis, although commonly encountered in the submaxillary, cervical, temporal, parotid regions and cheeks, occurs only infrequently in the mouth and pharynx. In the former situations, secondary extension with involvement of the ear and mastoid may take place and an abscess may rupture into the external auditory canal. In the mouth and throat, the infection is most often seen in the body and base of the tongue. The onset may be acute with severe, throbbing pain, local tenderness, general malaise and fever, as in pyogenic abscess, or insidious with slow progression during the course of several months. The latter is the rule in the absence of secondary infection. Frequently the tumor in the tongue appears on clinical examination to be a benign fibrous nodule or an infected cyst. Within the course of a few days, however, the process may flare up acutely and a large portion of the tongue may become involved. When the posterior portion of the tongue is affected, dysphagia may be so extreme that the patient is unable to swallow even fluids. If the abscess is not drained and if the nature of the condition is not recognized, spontaneous rupture with subsequent repocketing and extension

of the infection occurs. Ultimately, the entire tongue and floor of the mouth may become involved and the process may extend through and present in the submaxillary and submental regions. The diagnosis is based on a demonstration of so-called sulfur bodies or Actinomyces. Under intensive treatment with iodides, irradiation and drainage (or excision in cases with localized lesions), the prognosis is good depending on the situation, extent and virulence of the infection.

Blastomycosis, while usually a systemic or cutaneous disease, may be localized primarily in the mucous membranes of the mouth or throat. In these situations the lesions present much the same clinical appearance as those involving the skin, that is, elevated verrucous plaques which tend to clear in the center and spread about the periphery, where minute yellowish abscesses are visible with a hand lens. Blastomyces may be demonstrated in the purulent material expressed from these abscesses. The organisms, which appear as budding yeast forms, are also demonstrable in microscopic sections of the involved tissues. When the lesions occur in the mouth and especially in the larynx, the clinical picture may be very suggestive of the verrucous type of tuberculosis and only careful study of a biopsy specimen will permit differentiation.

Torulosis, a rare disease due to the pathogenic yeast, Torula, which closely resembles Blastomyces, most often involves the brain and meninges. A localized form of the infection occurs in the upper respiratory tract and at times in the middle ear. This probably precedes systemic invasion by the organism. Cases in which the infection was localized in the pharynx have been reported by Jones, Gill and others. While the disease in the brain and thorax is invariably fatal, the infection in these early cases was arrested by roentgen therapy and the administration of iodides. Jones made the interesting observation that, in half of the twenty-two cases reported up to 1927, the infection was apparently primary in the nose, throat, or ears. The causative organisms, torulae, are double-contoured, refractile and budding, and similar in appearance but smaller than the blastomycetes. They may be demonstrated in smears of the discharge or in microscopic sections of the granuloma. In the tissues, the lesions of torulosis, blastomycosis and coccidioidal granuloma are very similar and definite differentiation is largely dependent on cultural studies.

Coccidioidal granuloma, which is endemic in the San Joaquin Valley in California, often strikingly simulates blastomycosis and, like the latter condition, may produce oral and pharyngeal ulcers and granulomatous lesions. It is due to the Coccidioides immitis, a double-contoured organism similar to Blastomyces and Torula but much larger than either of them. It reproduces by sporulation, never by budding as do the last named, and it may be demonstrated in smears or microscopic sections treated with potassium hydrate solution.

Leprosy, another of the chronic infectious granulomas, is due to the lepra bacillus. In the early stages it commonly affects the external nose as a nodular infiltration which later extends to involve the nasal mucosa and the palate, pharynx and larynx. Ulceration promptly develops in the involved mucous membrane and the secretion covering these lesions contains numerous clumps of lepra bacilli resembling bundles of cigars. As the active process subsides, atrophy and cicatrization with more or less distortion occurs. The lesions of leprosy strongly resemble those of syphilis and tuberculosis and, as both the Wassermann and tuberculin tests are positive in cases of leprosy, diagnosis is often difficult. In addition, the lepra bacillus is, like Mycobacterium tuberculosis, acid-fast, but it is non-pathogenic to animals.

Thrush, or moniliasis, may produce cutaneous lesions, but it much more commonly involves the oral mucous membrane, either as an acute or a chronic process. The causative organism is the parasitic fungus, Monilia albica. The acute form of the disease occurs most commonly among suckling infants and malnourished children less than five years of age. It is also seen among aged or debilitated individuals, especially during the later stages of tuberculosis and other wasting diseases and during convalescence following severe abdominal surgical operations. The condition is characterized by whitish or grayish, curdy exudative plaques covering a varying amount of the oral mucosa. As a rule this can be wiped away readily and leaves an extremely tender, excoriated and often oozing surface. The intense discomfort often associated with the disease may interfere markedly with the patient's nourishment. The clinical findings are characteristic and the diagnosis is confirmed by demonstration of yeast forms and mycelium in films or cultures of the membrane. The condition can often be cleared up with a single, thorough, local application of 10 per cent formalin following cocainization.

So-called submucous or chronic thrush is a more deeply seated process than the acute form. It occurs almost exclusively in adults, many of whom are in good general health otherwise. It is characterized by the presence of whitish, thread-like lesions within or below the mucous membrane. The entire surface of the tongue, as well as most of the remainder of the oral mucous membrane, may be infiltrated by the process, and this same involvement may extend through a varying portion of the gastro-intestinal tract. When the process is well localized in the mouth, destruction of the areas with the actual cautery or with a fine diathermy electrode, followed by the application of a 2 per cent solution of gentian violet is quite effective in relieving the discomfort, although complete eradication of the process is uncertain. The exudative type of chronic thrush infection is at times extremely persistent and has in some instances appeared to be a definite etiologic factor in the development of carcinoma of the buccal cavity.

In the differential diagnosis of submucous thrush, linear leukoplakia, lupus erythematosus and lichen planus must be ruled out. Considerable difficulty is often experienced in excluding the last named condition, although the cutaneous lesions of lichen planus, which are frequently situated on the wrists and other flexor surfaces, are of help. The oral lesions of the disease may occur in any portion of the mouth and they commonly appear as grayish striae which often form a diffuse network and at times thickened leukoplakia-like plaques. Histopathologic studies frequently are necessary for differentiation. According to Montgomery, these reveal a more edematous and boggy epidermis in submucous thrush, also definite spores and mycelial filaments in the tissues. Lupus erythematosus of the lips often produces a peculiar "silvering" due to adherent white scaling.

A case of primary tularemic ulceration of the pharynx was reported for the first time by McGovern in 1936. This condition is extremely rare, but it must be borne in mind as a diagnostic possibility when patients present extensive ulceration of the throat. In McGovern's case there were large confluent ulcerations of the pharynx and the cervical lymph nodes were enlarged and tender. The onset of the illness had been acute, with chills, severe sore throat and fever up to 104° F. The diagnosis of tularemia was based on strongly positive agglutination tests. A 1 per cent mercurochrome throat spray gave prompt and marked relief and the patient recovered without complications.

The earliest manifestations of pemphigus are not infrequently encountered in the mouth, pharynx and larynx; rarely are they encountered in the nose. During the course of the disease the eyes are commonly involved and, at times, the ears as well. The characteristic primary lesions, bullae on a noninflammatory base, may appear in the mouth or throat some little time prior to their development on the external body surface. They are usually only a few millimeters in diameter but may range up to 1 cm. or more. They are extremely thin-walled and inconspicuous and usually rupture within the course of a few hours. Careful observation of the patient for several days frequently is necessary in order that these bullae may be detected. Following rupture the flaccid pellicle sloughs off, leaving a superficial painful ulcer which promptly develops a marked secondary inflammatory reaction. These ulcers heal slowly and, as they often involve a considerable portion of the mucous membrane of the mouth and throat, the patients frequently are extremely uncomfortable. Treatment has not proved effective in controlling the condition to date and in the severe cases the prognosis is fatal.

Bullous lesions in the mouth may also be produced by erythema multiforme. However, this condition is as a rule readily differentiated by the history of similar attacks, which disappeared spontaneously, the seasonal incidence in the fall and spring, and by the multiformity and distribution of the lesions, especially on the extremities. Various drugs may likewise cause lesions in the mouth that might be confused with erythema multiforme, among them phenolphthalein, antipyrine, acetanilid, quinine, and the bromides, iodides, barbiturates and others.

A group of cases in which there was permanent enlargement of the lips and face (reported from The Mayo Clinic by New and Kirch a few years ago) are of interest in this connection. The condition occurs most commonly in young adults and is preceded by periodic attacks of swelling which most frequently involve the lips and cheeks but may affect the nose, eyelids, tongue and soft palate. The swelling develops in the course of a few hours, often without premonitory symptoms, and requires two weeks or more to clear up. After a second or third episode the swelling persists between the attacks so that the affected parts remain diffusely enlarged and the tissues firm and inelastic. Frequently the acute attacks are accompanied by facial paralysis on one or both sides. This often clears during the interim between attacks. The permanent enlarge-

ment does not progress beyond a moderate degree, although it is often sufficient to produce grotesque disfigurement. It is always less than the size attained during the acute stage. The condition is not preceded by lymphangitis or erysipelas. Histologically, there is edema and lymphocytic infiltration only. The process has been controlled with injections of hot water or alcohol into the subcutaneous tissues and by external irradiation with subsequent plastic surgical procedures to shape up the parts if necessary.

Allergic reactions to a great variety of foods, certain cosmetics and clothing may be responsible for dermatitis of the external ear and for gingivitis and stomatitis in sensitive individuals. The offending irritant in such cases frequently can only be determined by careful observation and by sensitization studies.

Aspergillosis due to a group of pathogenic molds most commonly occurs in the external auditory canal and in old pulmonary abscess cavities. Varieties of the Aspergillus usually encountered are the niger, flavus and fumigatus. Of these, the last is the most frequent invader. The presence of cerumen favors the development of the fungus in the ear canal. Clinically the condition appears as a dark, moldy membrane which may produce only blocking of the canal with mycelium and resultant impairment of hearing, or there may be ulceration and suppuration of the wall of the canal or penetration of the membrana tympani and involvement of the middle ear. Rarely, the mycosis involves the mouth, nasal fossa, antrum, ethmoids, sphenoid sinus and orbit. Treatment consists in improving local hygienic conditions together with antiseptic applications, such as 50 per cent alcohol containing small amounts of bichloride of mercury and boric acid or mercuric iodide.

Furunculosis, herpes, eczema, seborrhea and erysipelas of the ear are familiar to all otolaryngologists and need only be mentioned.

Dissimilar metallic dentures in the mouth at times cause local irritation and even leukoplakia in some cases by virtue of galvanic phenomena. In my experience, however, oral lesions due to this type of reaction are rare.

Aphthous and herpetic stomatitis are also familiar to all and need not be discussed.

Leukoplakia may occur in the mouth, pharynx, or larynx but is most frequent in the buccal cavity. It is evidence of a defensive reaction on the part of nature in an attempt to protect the oral tissues from some form of chronic irritation. Tobacco is the most

common irritant responsible for it, but dental factors, habitual biting of the lips and cheeks, and hot and spicy foods may play a part. Several distinct types of leukoplakia occur, and their appearance and clinical behavior vary considerably. Among these may be mentioned: (1) the squamous type, which is the most common form; (2) the papillary type; (3) that in which atrophic syphilitic glossitis is the predisposing cause, and (4) that in which interstitial syphilitic glossitis is present. The squamous type may or may not be associated with syphilis. It is of no consequence unless it becomes thickened and leathery, in which event its removal by electrocoagulation or the actual cautery is indicated because of the danger of malignant change. Prior to the development of thickening, removal of the cause will usually be followed by spontaneous clearing of the condition. Papillary leukoplakia is a potentially malignant lesion, tending to develop into the so-called frozen-doormat type of low-grade squamous-cell epithelioma. It appears as a thick, tough, warty, insensitive area and, like other forms of leukoplakia, is often discovered accidentally by the patient. The form of leukoplakia developing on the basis of atrophic syphilitic glossitis is also very prone to undergo malignant change and areas of ulceration or thickening arising in it must be viewed with suspicion and removed promptly. The tongue in such cases presents a smooth, glistening, ironed-out atrophic appearance and traumatizes readily. Carcinoma developing on this basis is usually of the active, squamous-cell type. When interstitial glossitis is present, secondary lobulation of the thickened grayish tongue surface results.

Keratosis pharyngis is characterized by the presence of numerous grayish, whitish, or yellowish horny out-growths which appear most commonly on the faucial tonsils, but not infrequently on the lingual tonsils and, at times, on the nasopharyngeal adenoid pad and lymphoid tissue elsewhere about the pharynx. This condition, which is caused by Leptothrix buccalis, is often symptomless and frequently is discovered accidentally. When forcibly removed, the adherent horny protuberances leave a denuded, bleeding surface and tend to reform promptly. They are prone to disappear spontaneously in the course of time and treatment is usually not indicated. If the condition is causing appreciable pharyngeal irritation, electrocoagulation of the individual points is justifiable.

Black tongue or hairy tongue appears primarily as a black or brownish hairy patch on the posterior portion of the dorsum of

this structure just anterior to the circumvallate papillae, but the condition may involve the entire dorsum. Histologically, the long hair-like processes on the surface of the tongue originate from abnormal papillae in the epithelial covering. According to Andrews, the color is due to changes in the horny cells analogous to those seen in certain types of hyperkeratosis of the cutaneous surface or to pigment from substances taken into the mouth. A foul taste is at times complained of by the patient but, more often, there are no associated symptoms. Treatment is usually of little avail. The condition often clears spontaneously but it may recur.

Another form of pseudoblack tongue is caused by a fungus which grows as a mold on the dorsal surface.

A degree of pigmentation of the buccal surfaces and alveolar processes is at times seen normally. As a rule, however, pigmentation of the mucous membrane of the nose, mouth and throat is pathologic. It may occur in various systemic diseases, such as hemochromatosis, Addison's disease, and acanthosis nigricans. It may also follow treatment with some of the heavy metals, namely, silver, gold, lead, bismuth and mercury, and also accidental or occupational poisoning by some of these, or phosphorus and similar chemicals. The blue line of lead and bismuth intoxication extending along the gingival border is well known. Extensive, diffuse, deep pigmentation of the mucous membrane of the nose, mouth and pharynx is at times seen extending widely about the periphery of melanotic epitheliomas in these situations.

Hypertrophy of the lingual papillae, producing furrowed or so-called scrotal tongue; geographic tongue, burning tongue and increased accumulation of lymphoid tissue along either lateral lingual border posteriorly, often cause much concern to the patient as well as to the physician who is unfamiliar with these conditions. None of these conditions is of clinical importance, however, and no local treatment is required. Ample reassurance is essential as fear of malignancy often forms the basis for complaints in this connection. Burning tongue as a rule is due to a psychic disturbance.

Glossitis rhomboidea mediana designates a benign lesion of the tongue, often slightly elevated and of an ovoid or rhomboid shape, situated in the midline of the dorsum just anterior to the circumvallate papillae. The condition is usually symptomless and in most instances is discovered accidentally. It is frequently mistaken for carcinoma.

So-called Fordyce's disease is the anomalous presence of numerous enlarged sebaceous glands in the oral mucosa. These appear as minute to pinhead-sized whitish or yellowish granular nodules within the mucous membrane, most often on the posterior portion of the cheeks and in the lips near the angles of the mouth. The condition is not pathologic and no treatment is indicated.

Benign hypertrophy of the lymphoid tissue over the base of the tongue and along the lateral pharyngeal folds is at times pronounced and necessitates treatment. Electrocoagulation, with or without direct application of radium to the surface, and external irradiation, followed by the administration of iodides, is the most effective treatment.

Xanthomas of the pharynx and larynx are rare. Two cases were reported from The Mayo Clinic by Finney, Montgomery and New in 1932 and New subsequently reported two additional cases (1935). The etiology of xanthoma is not known, but an abnormality of lipid metabolism associated with some local condition is thought to be responsible. In some cases the condition is associated with diabetes mellitus. Multiple yellowish nodules or plaques of varying size and distribution may be present in the mouth, pharynx, or trachea. These may be of sufficient size and extent to produce marked hoarseness. The histopathologic picture is similar for all types of xanthoma. According to the foregoing authors, the process may be divided rather arbitrarily into an early or inflammatory stage, a tumor stage, and a stage of regression or fibrosis. The most prominent feature is the presence of the xanthoma or foam cells which with fat stains are seen to be laden with various lipids and which are apparently derived from the reticulo-endothelial system. "Touton" giant cells, characterized by a central circular arrangement of their nuclei and a peripheral zone of lipid-containing cytoplasm, are frequently seen, especially in the tumor stage. Treatment, aside from surgical removal of such tumors, is of little avail.

SUMMARY

Different dermatologic conditions affecting the ears, nose, mouth and throat have been reviewed. A group of these represent a purely local condition; more of them, however, are a manifestation of some systemic disease. They are of interest to the otolaryngologist not only because of the therapeutic indications arising in connection with the local pathologic changes, but also be-

cause of their diagnostic and prognostic features in general disease. Recognition is at times possible from observation of the lesions of the mucous membrane alone, but careful study of the cutaneous surface, various laboratory investigations and complete physical examination may also be required.

[Conditions mentioned above were illustrated by means of lantern slides.]

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Symposium on Dermatological Lesions
About the Eyes, Ears, Nose and Throat

* * *

CONSTITUTIONAL BACKGROUND, DIFFERENTIAL DIAGNOSIS, AND TREATMENT OF DERMATOLOGICAL LESIONS ABOUT THE EYES, EARS, NOSE AND THROAT

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I should like at this time to express my very great appreciation for the privilege first of listening to the excellent presentations of Dr. Von der Heydt and Dr. Figi, but more for the courtesy that you have extended to me in asking me to present the differential diagnosis, therapy and background of the various lesions around the eye, ear, nose and throat. I may say that this is a pretty large order in the time allowed; in fact, it is quite impossible to complete the subject as it has been announced.

Furthermore, I think it is proper to say at this time that for the most part lesions which occur in the locations under discussion are not lesions which are solely in these locations, but they occur there perhaps by predominance and often occur for the time being at least as the sole manifestations of generalized eruptions of one type or another, or are manifestations of a systemic disease.

I have selected from a very large material a number of lantern slides which perhaps will seem a little drab to you after seeing the beautiful photochromes of both Doctors Von der Heydt and Figi, but they will illustrate to you some of the more common lesions which have already been described, and I propose to tell you something about the differential diagnosis and how closely they mimic one another.

The differentiation of various lesions as they appear on the skin and mucosae is a study of minutiae. These lesions so closely simulate each other that even the most careful clinical diagnosis sometimes is impossible without laboratory aid.

I have a number of slides which I will show to you now, which will illustrate this point.

Dr. Figi has shown you some primary sores of the lip. I want to show you a few that are not as typical as these but after all it is not the typical sore which is important. The average chancre reaches its maturity in a certain length of time and sooner or later it is usually recognized by its clinical manifestations and differentiated from the conditions which it usually simulates. It is the unusual chancre, the one which does not reach maturity, which is a dangerous lesion, and it is well for us to know and remember that many chancres occur simply as minor erosions which last, instead of from three to four weeks, sometimes only a matter of hours and frequently a matter of a very few days and are passed off as fever blisters or canker sores, fissures, and the like, and their true nature is not recognized until after some serious accident occurs to the individual.

The characteristic of a chancre which makes it relatively easy to diagnose when it is a mature chancre, such as this one, is induration, and we must recognize the difference between induration, which is a cirmumscribed hardness, and infiltration, which is a hardness which is not circumscribed but is spread to the surrounding tissue.

I want to differ in one respect from Dr. Figi when he said these lesions often have a foul discharge. Ordinarily the discharge from chancre is serous and has no odor.

The etiology and background, of course, of this type of primary sore is usually an osculation affair.

One finds a great many bizarre types of chancres which are contracted in other ways than from kissing. For example, there are certain cases of industrial forms of infection in which glass blowers have infected each other. That is the so-called mediate type of infection which occurs from contact not from the syphilitic virus from another patient, but in which the virus is contacted from an inanimate object which has been in contact with another patient. Whereas any chancre which is seen on the lip usually has a background of exposure to another individual, it need not necessarily so happen. We have occasional cases where the clinical thermometer has carried a chancre from one individual to another; where a pipe or cigarette holder has carried it; there are a number of other bizarre types of infection. This one happens to be a garden variety of osculation sore. (Referring to slide.)

The most important differential point about any chancre is the satellite bubo which is present and continues for considerable time after the chancre undergoes either spontaneous involution or involution under treatment.

Here is a rather unusual primary sore. Nine-tenths of the sores that occur on the lips occur on the lower lip; it is a much wider surface for infection, much more labile, and much more subject to tiny fissures and cracks which permit the introduction of the spirochete. But this is on the upper lip. Remember, if you will, that it takes from three to four weeks for a chancre to develop and several days from its development to the point of its maturity, and therefore the patient here shown actually had syphilis long before the chancre could be demonstrated. There are numerous cases of infections having taken place accidentally or having been transferred from one individual in the incubation period of chancre to another; especially can this be the case in transfusion syphilis.

Here you see a slide representing an exception to the rule that ordinarily postulates the chancre as a solitary lesion. It must be stated that once a chancre has developed, another chancre cannot occur and does not occur in the individual and there is no contact infection from one point to another, but occasionally the syphilitic virus enters in two or more distinct places at the same time, and under those conditions a double chancre, a triple chancre, or even four chancres may develop. In this case there were two, and I have in my collection a case in which there were two on the lip and one on the tongue.

Here is a picture that I am very glad to show you because it is a chancre of the upper lip, and I am presently going to show you for differential diagnosis a gumma of the upper lip which is so much like it that it is almost impossible to make a clinical diagnosis without other data.

There is a gumma of the upper lip. Both the chancre and the gumma are definitely discrete, definitely, sharply demarcated; they are both obviously syphilitic to one who is familiar with lesions of early or late syphilis, but they do closely simulate each other, though there are no superficial criteria for differentiation. However, when the cases are carefully examined, in the case of the chancre there is a satellite bubo, in the case of the gumma there is not; in the case of the chancre you are dealing with a distinctly indurated lesion with little or no ulceration, but in the

case of the gumma there is ulceration and it is sharply circumscribed as you see here.

I have a collection of gummas which by predilection occur in women with occult untreated syphilis around the nose and mouth, and I propose to show you some of them.

Here is a very interesting form of diffuse syphiloma of the lip, which is the so-called late syphilitic lymphangitis. It looks a good deal like an ordinary lymphangitis except that it is extremely hard. It has a rubbery consistency, comes on rather slowly, and has no tendency to ulceration; it persists as a deep indurated lesion presenting this apron-like fullness of the lower lip, and is not a true gumma, but a diffuse syphiloma of the lip.

Here are two lesions which are extremely interesting and which are problems in differential diagnosis. The first one is a mere keloid of the lip, a solitary nodule; it looks a good deal like a chancre. On the other side you see a simple fissure or so-called fissured chancre of the lower lip without the characteristic raised papulation that most chancres have. The differential diagnosis in this case will not be difficult because in association with the keloid you have no lymphadenitis, no tendency to erosion, no serous discharge, and a lesion of some induration, as keloids have, but with no tendency to progression and no tendency to retrogression. The chancre, on the other hand, does not remain in situ long, even after it reaches this full size. Finally it disappears under treatment very rapidly. But if one saw this keloidal lesion at first glance, it would look a little like an involuting primary sore. The second lesion over here is a chancre occurring in a fissure which has not reached full size and its differential diagnosis depends first on the induration of the lesion, on the lymphadenitis, and of course all these lesions demonstrate their nature by careful dark-field examination.

Just to show you how these lesions simulate each other, here are two other slides showing simulation, one a cancer, the other a chancre. There are no definite criteria of age to depend upon. To be sure, one expects syphilis in the early years of life and cancer in the late years of life, but I have in my collection chancres occurring in a man over seventy-nine and squamous cell carcinoma occurring in young people before their twenty-first year. So age cannot be regarded as an absolute criterion for differential diagnosis.

The lesions are both solitary. The cancerous lesion is usually slow to develop, is definitely infiltrated instead of indurated, it

tends easily to bleed, it is slightly painful; whereas the chancre is quite painless, it is rapid in evolution, and the most important differential point is again in the presence or absence of a lymph gland involvement. In the case of cancer the lymph gland involvement is usually late and then the glands are usually small and rather shotty; in the case of the chancre the glands are inevitably involved early, are large, painless, and tend to disappear slowly after the primary sore has disappeared.

We have had some very interesting tongues shown to us by Dr. Figi. I will show you one that causes a great deal of dismay needlessly to a good many people. Patients will come to you and say they have a lesion on the tongue, and they frequently have a cancer phobia.

This is so-called wandering rash of the tongue, or lingua geographica. It is of practically no significance except that it causes a great deal of anxiety. Its etiology is not known. It occurs frequently in patients with mild gastro-intestinal symptoms, occasionally in those with chronic hyperacidity, for example. However, it occurs without any known cause and may persist for year after year. It behaves a good deal as though it might be a parasitic disease, but we have failed to demonstrate parasites in any case at all. There is no potentiality toward malignancy; that is, it is not a so-called pre-cancerosis, but may be a source of a great deal of worry to the patients who have it.

This type of Moeller's glossitis is of extremely great interest because it is usually indicative of serious systemic disease. It is found frequently in pernicious anemia, in cases of abdominal carcinoma, in gastric carcinoma, in severe anemias, and occasionally in primary blood dyscrasias. There is, as you see, a smooth atrophy of the tongue with a sensation also of burning. Patients will say that their tongue is sensitive to heat, to cold, to acids and certain foods; the presence of a smooth tongue in which the papillæ have disappeared, with a sensation of burning, should always lead to a careful physical examination of the patient, particularly of the blood to determine the presence of pernicious anemia, and a careful check-up of the entire patient for the demonstration possibly of malignancy.

Here is another not uncommon anomaly that leads to a great deal of trouble. Some patients will have this so-called scrotal tongue for twenty or thirty years and never know anything about it until some day they stick their tongue out and notice it is not like other people's and they come to you in fear and trembling.

Scrotal tongue is merely an anomalous condition; it is something of a nevus type of tongue, but a perfectly useful organ. If the patient does not become tongue conscious or alarmed by some person who is ill informed, if a dentist doesn't say, "There must be something wrong with your tongue, you must see your doctor," or if a member of the family doesn't frighten him about it, he may remain unconscious of it for life. A scrotal tongue is of absolutely no significance clinically.

On this side, however, there is an interesting tongue which is the analogue of the one Dr. Figi showed you. It is a tongue occurring in a case of lymphoblastoma or of a lymphomatous condition which we call mycosis fungoides. These lymphomablastomatous conditions include the leukemias, lymphosarcoma, Hodgkin's disease, and this interesting disease called mycosis fungoides. The bone marrow and the lymphogenous elements of the body, particularly the primitive lymph nodes and the skin, are attacked by this very rapidly fatal disease.

Lichen Planus. This is an actual photograph of lichen planus of the mucous membrane. That is also a cause of a great deal of apprehension, not only on the part of the patient, but on the part of the specialist physician. I have had a number of patients who have had lichen planus for years, referred to me for syphilis, and lichen planus has of course nothing whatever to do with syphilis. It is ordinarily found on the skin with associated findings in the mucosa. I was among the first to describe the solitary lesions in the mucous membranes. They may occur on the tongue, but more frequently on the buccal mucosa.

There is a suggestive background to lichen planus. Etiologically we do not know precisely what it is. We feel reasonably certain that it is not an infection, but we do know that it occurs in certain types of individuals; we know that it occurs rather frequently in patients who have been under severe mental strain, perhaps some severe illness in a member of the family or the death of a member of the family; then the patient breaks out with these lichen planus lesions. They occur on the body as flat purple colored lesions, particularly on the extremities, and associated with itching. On the mucosa they do not look like that at all; they look like a mosaic grayish inlay. The patient hardly ever feels them; they do not usually protrude above the mucous surface. They do look a little like syphilids but they differ definitely from them; the syphilids are raised, circumscribed, clearcut, or cleancut erosions or ulcers

of transitory character; the lichen planus is a mosaic which spreads and persists and is characterized by its persistence in the mouth.

One of the things that has induced a great many physicians into the belief that they are dealing with syphilis is that lichen planus is amenable to mercury treatment. The best way we have of treating them is by injections of mercury.

The black hairy tongue you see here I will only allude to a moment. As Dr. Figi said, in some cases parasites have been found. I have found in some cases the Aspergillus niger, the black Aspergillus. The people who most frequently have this are tobacco chewers, and it is not unlikely that the tar of tobacco smoke is very largely responsible for the color of the black hairy tongue. The hairs are not hairs at all; they are simply soft papillomata; they are hypertrophic infective lesions similar to the lesions of the so-called venereal wart or the fungating or villiform growth stimulated by mild forms of staphylogenic infection.

Lichen planus you see on the tongue here, also associated with a generalized eruption. It must be differentiated from leukoplakia, and is not difficult of differentiation because lichen planus ordinarily is not a hypertrophic lesion, whereas leukokeratosis is a raised white patch due to increased epithelial growth.

Going back again to the chancre, the chancre of the tongue on the tip is usually of the same etiology as the chancre of the lip. It may be due to direct contact; however, it may again be due to mediate forms of infection where some object is placed in the mouth or near the tongue which previously has had contact with the syphilitic individual. One of the most interesting cases I know of, of which I have seen two examples, was a nursemaid who was feeding hot cereal to an infant, and wanting to be perfectly sure it would not burn the child she would first taste the spoon or put it in her mouth and then put it in the infant's mouth, and a resultant chancre occurred in this way. That is a form of mediate infection.

This is syphilitic interstitial glossitis, a smooth atrophy. That is the analogue of what you find in liver syphilis, in the so-called hepar lobatum. This is the result of an old gumma, or multiple gummas of the muscularis. The tongue is shrunken and furrowed and has scars in it; it is a form of interstitial deep-seated syphilitic glossitis, absolutely characteristic when you see it, and puts you in mind of an old hepar lobatum.

Here is another one, the one on this side with leukoplakia and cancer. What is the relation of syphilis to cancer when it occurs on

the tongue or the mucous membranes? Billroth was authority for the statement that the majority of cases of cancer of the mouth, of the lip, of the tongue, could be ascribed to syphilis, and yet there is nothing whatever in the syphilitic lesion per se which predisposes to cancer. However, the syphilitic residua, such as the leukokeratosis or leukoplakia, are breaks in the continuity of surface epithelium which are subjected to physiologic trauma and occasionally to pathologic trauma such as smoking. Under those conditions the leukoplakia must be regarded as a pre-cancerosis. Leukoplakia is not invariably an old syphilomatous lesion or the result of one, but it very frequently is; that is to say, the old mucous patches, the old interstitial glossitis, leads to a change of epithelial structure. The process of keratization is incomplete and you have a nucleated horny layer on the tongue where nuclei should be absent, a rapidly developing epithelium in which the nuclei are preserved, and this change in epithelial development may lead to metaplasia. Malignant degeneration probably occurs in old syphilitic lesions not because they are syphilitic but because they are pre-cancerous lesions.

This is a leukoplasic tongue in which there is a negative history of syphilis, and on the other side one in a syphilitic individual in which malignant degeneration has already taken place in a leukoplakia.

Here is a very markedly hypertrophic leukokeratosis of the tongue. I don't think that these which are located in the center of the tongue are so apt to undergo malignant degeneration as those which occur on the side, but this sort of lesion, as Dr. Figi has suggested, should be destroyed long before it becomes a menace. The scar tissue resulting will not be covered by epithelium which is too rapidly developing and resulting in so-called parakeratosis, which is definitely pre-cancerous.

Tuberculosis of the tongue we do not see so very commonly. It is very seldom a primary affair; it is usually the result of very severe pulmonary infection, and not infrequently an end result or terminal stage late in tuberculosis of the larynx. It is usually a sputum infection of the mucosa, and for that reason it ordinarily has a rather bad prognosis. The only ones which have a fair prognosis are those in which there is an endogenous tuberculosis, in which lupus occurs first on the outside and then extends in to the lip. Those on the tongue or buccal mucosa are very serious and are characterized by pain, by their irregular fissured undermining appearance.

A differential diagnostic point of importance is the presence of minute white points on the edges of the sinus tract or ulcer, which are actually solitary tubercles. The diagnosis is easy, too, because they are usually patients who are dying of tuberculosis.

Here is a replica of what Dr. Figi showed you in actinomycosis of the tongue, which is not so bad an infection as I once believed it to be. We collected a number of these cases from the literature, and these are the cases actually where the actinomyces or ray fungus is picked up in the field from a straw or a bit of hay; that is, they are the true analogue of lumpy jaw of cattle. Most of our cases of human actinomycosis are not cases of lumpy jaw. They occur elsewhere, in the abdomen, on the back of the neck, on the hand; a few of them do occur on the gums, and those are the cases in which the source of the infection is the same as with cattle. This lesion was very successfully treated by excision and by full doses of iodide and X-ray. Iodide and X-ray are, for most cases of actinomycosis, about the only things we have besides surgical removal of the tissue.

There is an actinomycosis too well developed to mistake. This is a true case of lumpy jaw. As in cattle, lumpy jaw is a very painful swelling at the side of the face, usually reddened in color, with numerous sinuses leading to bone, and pressure upon the lesion usually leads to the extrusion of a thin seropurulent material from which small yellow sulphur granules can be picked up. They are about the size of a pinhead, and placed upon a slide they will show you practically enormous colonies, pure colonies of the ray fungus.

The bone here was not extensively involved. This case was successfully treated with full doses of X-ray, Lugol's solution externally, and iodide internally in very large doses.

Here is an interesting case. Dr. Von der Heydt showed you ringworm of the eyelids. I have never seen just the sort of case that he showed of simple tinea or simple epidermophytosis of the eyelid; it is rare.

This, however, is a case of one of the most serious forms of skin fungus infection that we know, namely, favus or tinea favosa. This man has favus of his eyebrows and eyelashes and scalp. This is a form of infection that involves lower animals, particularly rats and mice. It is easy to diagnose because it produces a sort of sulphur yellow crust which has a distinctive mousy odor and invariably produces definite scarring. It requires many, many months and frequently a few years to rid these patients of the infection,

and it is an extremely unfortunate infection to anyone who has it so far as end results and scarring are concerned.

This slide shows a very interesting example, again, of what Dr. Figi has shown you. This is moniliasis or oidiomycosis of the face, and in a child it almost invariably has this background: the infection is usually a lower bowel infection. One finds enormous numbers of Monilia in the bowel; the child reinfects itself, its fingers, from its stool and then infects its mouth. It is a vicious cycle that goes around for months and months, in this case for two years, an extremely difficult disease to handle. As the individual gets older and can cooperate a little bit better, can control the reinfection, and can be made to maintain an absolutely strict regimen, these cases occasionally can be most successfully treated, and they must be treated by very careful treatment of the flora of the intestine itself as well as by local treatment of the mouth and nose. The mouth in this case is very extensively involved, also the eyes and the intestinal tract.

Here we have a dangerous lesion of secondary syphilis, one that shows itself as very characteristic. Such lesions quite often are mistaken because of their relatively benign appearance. The mucous patch is nothing but a papule on the mucous membrane. The papular syphilid of the skin is not a dangerous lesion as far as anybody else is concerned because there are no organisms on the surface of it until it becomes an eroded moist lesion. However, when it occurs on the mucous membrane it immediately becomes the most serious type of lesion, it swarms with organisms, it has a tendency to disappear and to reappear, and it is not infrequently encountered one, two and three years after infection. The recurring ones are the ones which are so apt to cause serious trouble to the patient and the doctor and to others, because they are not accompanied by an exanthem; they are recurring conditions, so-called mucocutaneous recurrences.

Here is a syphilitic mucous patch which might very well have escaped attention if one had looked rather casually at the mucous membrane, because the under surface of the upper lip is sometimes overlooked. It emphasizes the importance of examining every corner of the mucous membranes of the mouth because it is in the unexpected places that lesions of this type are found.

The next slide shows split papules at the angle of the mouth, the most dangerous form of lesion syphilis can produce so far as the patient and others are concerned. I have seen two infections in dentists and one in a dental surgeon from lesions of this char-

acter when they were not recognized. These are usually thought to be impetiginous or streptogenic infections of the angle of the mouth and are sometimes not infrequently mistaken for simple herpes. They swarm with organisms, they come and go, and you can see how easy it would be for one to become accidentally infected from a lesion of this sort in a patient with a syphilis unrecognized and untreated. Any lesion which is a papule at the angle of the mouth should always be regarded with suspicion until you can be sure that it is not a recurrent syphilid papule of this type.

These lesions do not necessarily occur in both angles; they may occur on one side, disappear, and come back over and over again. For example, a patient goes to the dentist's chair, has a protracted treatment in which a rubber dam is used, the mouth is placed on a stretch, there is slight trauma, and in a week or so he presents himself with a split papule.

You will see here that careful examination will show that these are definitely circumscribed indurated lesions. They should be dealt with with suspicion because of their actual morphology.

Here next are the mucous patches on the tongue, on the left side, which show you how definitely circumscribed and how clean-cut and clear the papules on the tongue are. They are not irregular, they tend to be definitely regular, and furthermore they do not coalesce; they tend to remain discrete. They have a grayish pellicle that can be removed with a great deal of difficulty. The discreteness and the grayish color of the papule should always arouse suspicion of their nature. They do not necessarily accompany the exanthem of early secondary period. They may come back three or four years after the infection.

Here are the lesions of secondary syphilis which tend to predominate occasionally around the face. I have shown this one particularly because this is a papulopustular syphilid with lesions around the eye, also lesions around the mouth, and they are definitely circumscribed. They do not itch; they have very little tendency to any symptomatology, and they may come and go.

Here is a pustular syphilid which is of particular interest to ophthalmologists. This lesion looked a good deal like the lesion of a pustular rosacea. The lesions are indurated, they come on rather quickly and do not coalesce.

I am showing you this case, which is a large lenticular syphilid, because of its close resemblance to leprosy. When I saw this young woman she had lesions on her face and forehead. The sites of

election for nodular leprosy are on the eyebrow, the ears, the bridge of the nose, and the cheeks. These are rather soft, brownish lesions. I have taken these simply to show you, together with the next slide, how closely they may simulate leprosy.

This slide is a picture of a case of leprosy which we picked up right in the State of Michigan, and you see that you have here the typical leontiasis or leonine facies, with lesions tending to occur in the eyebrows, on the nose, the mouth and the lips, but one can see leonine facies in a modified form in the lenticular syphilid which you have just seen.

In regard to tuberculosis around the face, there are a great many varieties of lesions. Briefly, they are either endogenous forms of tuberculosis of a very mild type which occur in this form as lupus vulgaris progressing very slowly and gradually producing atrophic scarring.

Then there is a primary tubercular complex in which an actual tuberculoma may occur on the skin from contact with tubercle bacilli from the mouth.

Next there is a warty form of tuberculosis, a tuberculosis which occurs from contact with the tubercular material or sputum and which may occur anywhere on the body where it is rubbed into the surface of the skin and produces a wart-like lesion. That is the so-called prosectors wart or anatomical wart and occasionally it spreads out to form a very large verrucous lesion.

Then there is an exanthematic tuberculosis which occurs as miliary tuberculosis of the skin. These lesions of tuberculosis of the skin have completely changed the concept of miliary tuberculosis. Miliary tuberculosis formerly was believed to be uniformly a rapidly progressing fatal disease. Now we know there are cases of miliary tuberculosis in which the skin is the site of thousands of minute tubercules which run a very mild course and gradually undergo a lysis and disappear. I am going to show you some of those in just a moment.

Both Dr. Figi and Dr. Von der Heydt showed you examples of rosacea. I am particularly interested in these because we have recently described a rosacea-like form of tuberculosis. The patients, so far in all of our cases, have been women, young women, who have a reddish congested face, a reddish nose and forehead, a few little papular and pustular lesions, and they don't get well as the ordinary case of rosacea does with local treatment.

The patient with a true rosacea frequently has a background of gastric anacidity or hyperacidity; they are dyspeptic individuals.

The patients with rosacea-like tuberculosis do not have this background. When you eliminate the red color from these patients' faces by pressing on them with a glass slide you can see that the face is studded with tiny little lemon yellow or apple jelly nodules. In such cases there may be an old glandular tuberculosis, frequently in the neck, an old inspissated gland which may be calcified, and which is occasionally seen in the X-ray. For reasons unknown the glands break down and there is an immediate diffusion into the blood stream of tubercle bacilli of low-grade virulence which lodge in the face. Those cases are very successfully treated as far as the lesions are concerned with the water-cooled lamp. They are given several blistering doses and the lesions disappear, but they may recur, unless the focus is found and destroyed.

This slide shows a form of rosacea-like tuberculosis. Here is another one that looks like rosacea. Each one of these minute lesions is a solitary tubercle. In two of these cases we could actually find evidence of old tuberculosis in the glands of the neck.

Here is the third one, the most characteristic one of all. This young girl gave a history of pulmonary tuberculosis in childhood. This slide is an example of miliary tuberculosis of the face, so-called tuberculosis miliaris facies. The extraordinary predilection of this disease for the face is as yet an unexplainable phenomenon.

This slide shows the patient whose tongue you saw with mycosis fungoides which you remember is a lymphoblastoma arising in the primitive lymph nodes of the skin and invariably is a fatal disease. The initial lesions begin as very benign looking plaques not unlike those of dry eczema. Over a period of months and occasionally years, they become infiltrated and form flat plaque-like lesions. Then with the onset of itching, they develop into smaller and larger tumors, which become necrotic and extremely foul. While the slide that you see shows lesions only on the face, the entire body is usually involved in this extraordinary malignant process.

This next slide shows you an interesting case of lymphatic leukemia with metastatis leukemic lesions in the neck, under the eye, and around the mouth.

Next you see an excellent picture of blastomycosis. In this you will note the sharp demarcation and the warty hypertrophic character of the lesion. If one squeezes this lesion, minute droplets of pus can be extruded, and microscopic examination of the material shows the double refractive bodies, the blastomycetes, which Dr. Figi's microphotograph showed so clearly.

Blastomycosis is not necessarily systemic. The cases where the skin only is involved are curable by thorough curettement, the application of iodine solutions locally, X-ray treatment, and full doses of iodide internally.

Here you see another blastomycosis. This patient left our hospital some six months ago, having to all intents been cured of the most extensive case of cutaneous blastomycosis that I ever saw. His lesion now is represented by a simple superficial scar. The cure was effected by X-ray, curettement, and potassium iodide.

Next I should like to show you the extraordinary gummas around the nose and upper lip that I mentioned before as having been seen almost exclusively in women. We have a fairly large collection of lesions of this type occurring in unsuspected occult syphilis in women. As you see, all these lesions have the common characteristics of being serpiginous in outline, sharply demarcated, occurring about the nares, and a characteristic advancing border. Most of them are treated and diagnosed as some form of tuberculosis.

Next is a destructive lesion in which not only the nose is involved, but also the eyelid and forehead.

Next a gumma of the side of the face involving a portion of the parotid gland, very interesting from the diagnostic standpoint in that it resembles somewhat an actinomycosis.

Here you see a gumma of the lymph glands of the neck in which differentiation from tuberculosis was necessary. Superficially the cases resemble each other, but the gummous form tends much more to destruction of the entire gland, and its extrusion as a slough with a gummous ulceration of the skin, whereas in tuberculosis the destruction is much slower and leads to sinus formation.

Now I desire to show you a gumma of the side of the face, and immediately following it a cancer of the same location, to illustrate how these conditions may simulate each other. The gumma is a solitary one of the cheek. It began as a small nodule, rapidly became infarcted, sloughed out, and left this punched-out circumscribed ulcer. On the next slide you see a lesion so much like it that at first glance it might be mistaken for another gumma. This, however, is a carcinoma, and if one looked at the edge of the lesion it would be noticed that it had a pearly raised everted border of peculiar translucency, that it tends to bleed easily, and that it is firmly fixed to the underlying tissue. The gumma, on the other hand, is a perfectly clean punched-out ulcer, so that on close in-

spection these lesions really differ and resemble each other superficially only in that both are round, solitary lesions in the same location.

The next slide shows a gumma of the nares which I have brought to illustrate how it may simulate the rhinoscleroma which Dr. Figi showed you. The gumma that you see here does not extend far up into the nares. It has produced some tumefaction, but it is distinctly a destructive, sharply limited process. Here is a case of rhinoscleroma which may be said to resemble it. You will notice here that there is no sharp outline, that the nares are filled with a hypertrophic granulomatous tissue in which destruction is not a characteristic feature.

I should now like to turn to eruptions around the face, neck, nose, eyes, and mouth, which are due to drug ingestion. Of these, phenolphthalein stands out as one of the greatest offenders. It is the basis of over a hundred forms of laxative handed over the drug store counters. In predisposed individuals phenolphthalein produces an extraordinary eruption. As you see here, this patient is covered with large pigmented lesions. These are persistent for many months and occasionally years, and result following urticaria-like lesions due to phenolphthalein. We are seeing more and more of these and more different forms of phenolphthalein eruptions. They not only occur as these brownish pigmented macules, but can actually produce blisters and erosions in the mouth and on the tongue and on the genitalia. The therapeutic test of such suspected cases, giving them small doses of phenolphthalein, is a very striking phenomenon.

The next slide will show you a bromine exanthema. Bromine will occasionally produce very serious lesions, and even in very minute doses can produce extraordinarily large nodules. The eruption which I am showing you in this young baby occurred as a generalized bromoderma from a single dose of bromoseltzer that the mother took while she was nursing this baby.

Here is another bromoderma in a patient who took a bromide for nervousness. There is some slight resemblance of these lesions to nodular syphilids, but they have not the sharp cleancut outlines of syphilitic lesions, and have a dirty brownish crust which is extremely characteristic of the bromide eruption.

The next case is one of iododerma which might readily be mistaken for the slide which I showed you of mycosis fungoides. There are a few cases on record where sensitiveness to iodine has led to fatal cutaneous disease.

The next slide illustrates a most interesting condition which was mentioned by one of the previous speakers, and is an example of acanthosis nigricans, a most unusual pigmentary disorder occurring around the eyes, the mouth, the neck, the axillae, and the genitalia in the form of a brownish-black hypertrophic lesion. Cases usually begin as a faintly brown color, the epithelium becomes heaped up, velvety in appearance, and then the color advances to dark brown and occasionally to black. This disease has a most interesting background. The majority of the cases are associated with some form of abdominal carcinoma, perhaps most frequently of carcinoma of the stomach, although cases have been described in which the neoplasm occurred in the adrenal, the rectum, the intestine, and the prostate.

I am showing you next a slide as an extreme example of freckling. Although this seems common, it nevertheless has a very interesting background of what we know as photosensitivity. The freckle is a frantic effort on the part of the skin to filter out injurious rays of light in patients who are not provided with adequate pigmentary protection against such rays. As you know, freckling is particularly common in redheaded people and blonds, and these people are much more susceptible to light than brunette types. Over-exposure to the X-ray can produce in brunettes, as well as blonds, freckles. A more advanced degree of such photosensitivity leading to freckling I will show you in the next two slides. Here are children with a congenital photosensitivity so great that they developed not only freckles, but the changes that we associate with senility, keratosis, and continued exposure leads these keratoses to a malignant degeneration. This disease is known as "xeroderma pigmentosum" and the unfortunate light-sensitive individuals of the type usually die before they reach puberty.

The next slide shows you a xanthomatous lesion which Dr. Figi called to your attention, and Dr. Von der Heydt mentioned as occurring around the eyelids. This young girl had xanthomatous lesions all over the body, varying from small pinhead lesions to those as large as a small plum. Xanthomas of this type are expressions of a most extraordinary disorder of lipid metabolism, occasionally limited to the cholesterol, but not necessarily so. In some instances the blood will be found to have a very high cholesterol content, and in others the fatty acids and neutral fats seem to be the disturbing factors. The girl that you see in this picture actually died of a xanthomatous lesion on the mitral valve. Patients with this disease are unable to metabolize fats properly, and

deposit them in the skin and in various parts of the body. If they are placed on a low maintenance diet they withdraw these fats, and if the maintenance diet is then withdrawn the fats are again thrust out into the tissues.

The next slide is an example of one of the cases that Dr. Figi showed you of erythema multiforme. As you see, there is a marked involvement on the skin as well as about the mouth and nose. The lesions on the skin have a characteristic round bull's eye appearance, the so-called erythema iris, and when these lesions are present they present no difficulty of diagnosis.

When bullous erythema, however, occurs in the mouth alone it is almost impossible to distinguish it from that fatal bullous disease that we know as pemphigus. Time and the development of other lesions are the only deciding factors.

The next two cases are those of pemphigus, of which there is no more fatal disease and no one more difficult of understanding. The blisters usually occur first in the mouth and for this reason patients frequently consult the otolaryngologist or the dentist. Many of them are diagnosed and treated as cases of Vincent's angina. Recovery from the first lesions is not unusual, but they invariably relapse and are accompanied ultimately with blisters of various parts of the body which gradually spread and ultimately lead to death. I have recently looked up my own experience with this extraordinary disease, and find that we have had eighty cases of pemphigus in my service during twenty-six years. Of these, one hundred per cent ended fatally within a few months.

Occasionally cases with bullous erythema may be seriously ill with the disease and resemble pemphigus in this way. They present the picture of mild sepsis and indeed are examples of mild septic infection. Except for the fact that cases of pemphigus show a marked predisposition for those of the Jewish race, and particularly of middle-aged patients, we know nothing of the further background of this disease. There is little evidence to support it as an infection. It is afebrile. There is no immunity or contagion and no evidence of infection at the post mortem. There are no visceral lesions of any kind except those associated with long standing cachexia. My own feeling is that this is a form of trophic disease, although there is no definite evidence to support this view. It is interesting, however, that this connection with blisters is frequently found in other trophic diseases, or in diseases in which nerve tissues are injured. Thus we find them in herpes zoster, in leprosy, and occasionally in syringomyelia.

Section on Otolaryngology

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HEARING AIDS*

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RECENT improvements in electrical hearing aids released with confusing frequency by competing manufacturers have imposed on the otolaryngologist new responsibilities and problems. His aurally handicapped patients confidently look to him to prescribe or give specific help in the selection of hearing devices best adapted to their individual requirements. To render this service satisfactorily requires a certain amount of elementary knowledge of construction, performance and limitation of these instruments.

To make more clear some of the physical features involved in the application of hearing aids Dr. Hartig presents (with demonstration) some of the acoustic phenomena concerned in the problem.

DISTORTION

In the normal process of communicating ideas by speech there are several indispensable elements, namely the speaker, the listener and a path of sound transmission. To insure a high degree of understandability each element of the combination must function properly, that is:

1. Speech enunciation must be clear and loud.
2. The listener's hearing must be normal.
3. The path of sound transmission must be free from echoes, reverberation, interfering noises or distortion.

The several elements involved are shown diagrammatically in Fig. 1.

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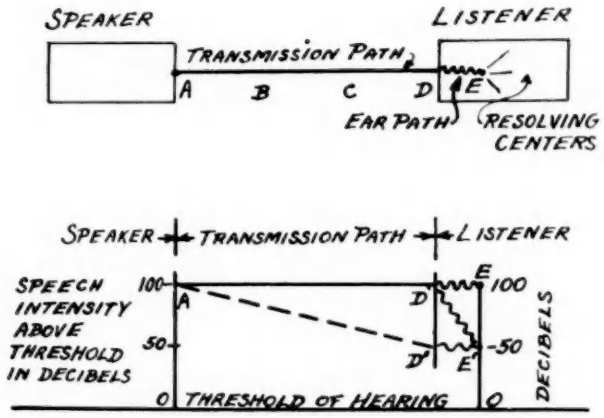

FIG. 1 — (Newhart and Hartig)

Part of the transmission path lies within the listener, as shown by DE. In the lower part of Fig. 1 is shown diagrammatically how the loudness level of speech may vary from the speaker to the listener.

Now suppose the sound intensity is insufficient for the listener. This may be due, as shown diagrammatically above, to either of two causes:

1. Loudness loss in the transmission path.
2. Hearing loss in the listener.

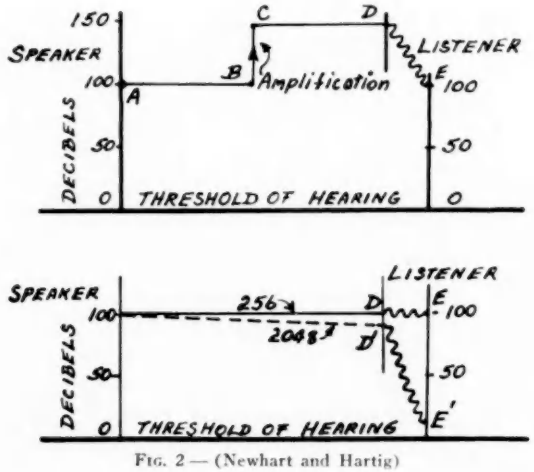

FIG. 2 — (Newhart and Hartig)

To remedy the condition of insufficient listener intensity, amplification may be resorted to with the result shown in Fig. 3.

Thus far we have assumed that speech was transmitted as a whole and that all of its parts were transmitted equally, but in reality the phenomenon is more complicated, due to the fact that speech is not like a simple tone but consists in its most important range of a band of tones ranging from 100 to 4000 vibrations per second. If for example we take two tones, 256 and 2048 vibrations per second, a possible vibration condition is that depicted in the lower diagram of Fig. 2 in which the listener's ear has no loss for 256 but has a 90 decibel loss for 2048. Now we may inquire how does speech distorted in this fashion sound to the listener? To find out we need only cause the same kind of loss in transmission between the speaker and the audience (which is

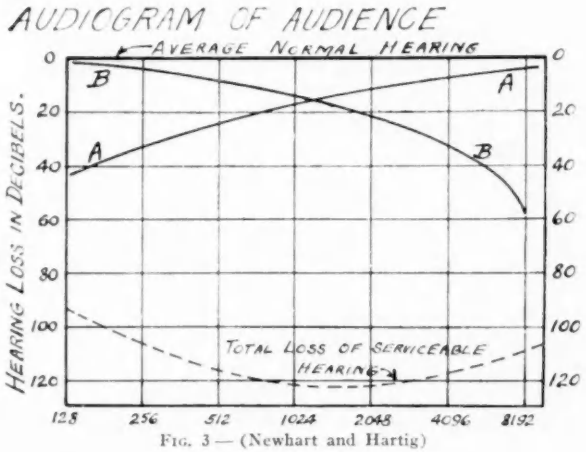

FIG. 3 — (Newhart and Hartig)

assumed to have normal hearing) as the person with defective hearing has in his own hearing mechanism. By means of the loud speaker system before me and a distorting network, the audience can be made to have effectively the audiogram shown in Fig. 3A. (The speaker then spoke several test sentences over a special loud speaker system.)

You will observe that two things happened when the audience was afflicted as indicated by audiogram A. First, the loudness of the sound was greatly reduced; second, the character of the speech sounds was greatly altered. But when the amplification was increased speech was still clear and understandable. Appar-

ently then the ear which is defective only by reason of a non-uniform conduction loss, is capable of accommodating itself fairly well to this type of speech distortion, provided the speech sounds are loud enough.

Similarly the audiogram of the audience can be made to have the hearing loss characteristic shown in Fig. 3, audiogram B. (Demonstration over loud speaker system.) Here again I believe you will agree that the speech distortion, while severe and of a different character than 3A, can be understood fairly well. It should be emphasized, however, that distortion of the kind produced is similar to a conduction loss in the patient. Obviously a hearing defect caused by a failure in the resolving powers of the ear cannot be simulated in a normal ear by external distortion.

But there is still another type of external speech distortion of an altogether different kind and which has the property, not of subtracting from the normal speech sounds but of adding other noise components. These noise components disturb the listener so that even though the wonted speech sounds are all present, the noise sounds obscure or mask them. It is a very common fault of electrical hearing aids which have carbon microphones. This type of distortion will be illustrated by amplifying the speech sounds as they emanate from one of the cheap and unacceptable carbon microphone hearing aids. (Demonstration non-linear distortion.) You will all agree that speech sounds as garbled as these, are difficult to understand and nerve-wracking to listen to.

It should be emphasized that the carbon microphone hearing aid you have just listened to is an exceptionally poor one. However, some effect of this kind is present even in the best of the carbon type aids. On the other hand the vacuum tube hearing aids with crystal or condenser microphones when properly designed are comparatively free from this kind of distortion and it is because of this that they give superior results.

In prescribing hearing aids individuals are classified on the basis of the degree of their hearing deficiency and the preponderance of a conductive or perceptive loss. Persons whose loss is moderate and fairly uniform through the speech range are easily fitted with a carbon microphone aid. Those who have a severe loss, especially for the higher frequencies present a more difficult problem.

The information afforded by an audiogram for both air and bone conduction is invaluable. This information may be obtained with less accuracy by time-consuming tuning fork tests. The

audiogram definitely indicates whether an air conduction or a bone conduction receiver should be used. It shows the degree of amplification required at different frequencies to compensate for the patient's deficiencies and whether his needs can be satisfactorily met by a carbon microphone instrument, with or without an amplifier, or if his loss is such as to require a vacuum tube aid with its still greater amplification and fidelity.

The matter of fitting hearing aids by closely matching the audiogram at many points as at octave letter intervals by selective amplification as has been proposed, is a requirement which, to many, seems unnecessarily severe. Further scientific investigation will decide this question with certainty.

Improvements in the range and quality of the performance of hearing aids, the development of selective amplification and the recent perfection of truly wearable vacuum tube instruments has increased greatly the field of application of electric hearing devices. Until lately, because of their limitations, the average person could not tolerate a carbon device unless his hearing loss in the better ear exceeded 35 to 40 decibels. Today many of the improved carbon instruments and the wearable vacuum tube services yield such relatively excellent performance that many persons whose loss is only 25 to 30 decibels use them with great comfort and satisfaction. It should be noted also by physicians and educators that many young children with a hearing loss so great that it prevents the acquisition of normal speech, can now learn to talk like normal hearing children by using a modern hearing aid at home and in the school.

Credit should be given to the manufacturers of hearing devices for two notable contributions to the cause of better hearing:

1. By greatly improving the performance of their products, it has been made possible for a constantly increasing number of persons to successfully supplement their defective hearing and insure their economic and social status.
2. Their extensive advertising of better hearing devices has indirectly served to stimulate wider interest in the problem of conserving the hearing and has overcome largely the existing prejudice against the wearing of a hearing aid and the employment of those having a handicapping hearing loss since they know it can be compensated by the use of a modern hearing device.

The American Medical Association, through the Council on Physical Therapy, of which Mr. Howard A. Carter is Secretary, and its Committee of Consulting Otologists, of which Dr. A. A. Hayden is Chairman, is generously rendering a valuable service in a serious attempt to formulate specifications for minimum standards for the construction and performance of hearing aids. Already it has raised to a higher ethical level the advertising, distribution and servicing of these appliances. To date the products of five of the larger manufacturers have been approved by the Council as "acceptable," are advertised in the Journal of the American Medical Association, and can be safely prescribed by the otologist. It remains for other less widely known ethical makers of reliable instruments to submit their aids to the Council. If these prove acceptable their makers and the public will be protected against the unscrupulous, high pressure salesmen of inferior and often worthless aids.

Because of the rapidity with which improved models with appealing features are released, on principle, it is unwise for the otologist to prescribe or recommend the device of only one manufacturer to the exclusion of others. The patient should be allowed the choice of several aids of different makers. Otherwise he may be dissatisfied and discredit the recommended aid, the agent and the prescribing otologist. In the last analysis, the ultimate test of the efficiency of a hearing aid is the easily made intelligibility test. Every otologist should be familiar with its application. Comparative intelligibility tests of different aids must be made under identical conditions, the person making the test having no financial interest in the sale of any instrument.

The advent of the audiometer and the development of selective amplification have made it possible for the distributors or salesmen under the title of "consultant in hearing aids" or "audiometrist" to become a disturbing factor in the close relationship between physician and patient. The makers of "accepted" hearing aids insist that their representatives be well-trained and instruct them to limit their efforts strictly to furnishing the hearing device which will best meet the needs of each prospect. They are urged to avoid the temptation to diagnose or to suggest the treatment of ear diseases, in violation of medical practice acts in force in all states. Nevertheless, the opportunity and temptation to violate this principle exist and endanger the patient's best interests and jeopardize the prestige of the medical profession.

The imposing equipment of the lay "consultant," including an audiometer, a master hearing aid and a noise-proof testing booth; the interested, confidence-inspiring manner with which he does his work, and his understanding of the psychology of the handicapped patient all tend to convince the latter that he is in the hands of one better qualified to solve his hearing problems than the physician or otologist he may have consulted.

To meet most effectively this serious situation it is necessary that the otologist so equip himself and render such superior service that he will not suffer by comparison with the hearing aid distributor or lay "consultant."

The otologist in referring persons to the distributor of hearing aids should impress each patient with the fact that the use of a hearing aid will not prove the complete solution of his hearing problem, and that he should make every effort to preserve his residual hearing by keeping in close touch with an understanding, interested otologist or physician. In many cases, by close supervision of his health, much may be accomplished to arrest or retard further hearing loss. With a mastery of lip-reading and acquired skill in the use of the best hearing aid he can procure, the aurally handicapped person can long retain his contacts with his fellows and insure his economic and social security.

CONCLUSIONS

1. It is an important function of the otologist to help his aurally handicapped patient obtain the hearing instrument best suited to his individual requirements; to protect him from the exaggerated claims and high pressure methods of unscrupulous salesmen, particularly those selling inferior, second-hand, obsolete or worthless devices; and to insure to him the privilege of chosing a hearing aid from among the accepted models of different manufacturers, provided that in their performance they meet his needs with equal effectiveness.

2. It is important that otologists be well equipped both as to their instrumentarium and technical knowledge, that the referred patient will not conclude that the hearing aid representative is better prepared to serve him than the medically trained man.

3. The medical profession should encourage the makers of high grade hearing aids to submit their products to the Council

on Physical Therapy of the American Medical Association for acceptance and should cooperate with them in their endeavor to distribute and service their devices according to the highest ethical standards.

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DISCUSSION

Dr. Austin A. Hayden, Chicago: Dr. Newhart's excellent paper and Professor Hartig's discussion, of course, are directed to better hearing for those who do not hear well, for the preservation of hearing or improvement of hearing for the defective ears and for the conservation of the hearing of everybody.

In discussing hearing aids, it seems advisable to me to review what has preceded the hearing aids that have been discussed this afternoon. Remember, all of this goes back to 1872 when Alexander Graham Bell devised the very simple hook-up of receiver, transmitter and battery for the ears of his own mother. It is a strange thing that almost fifty years passed before any substantial improvement was made on it and that was the adoption of the stethoscope type into a molded ear piece, which occurred in 1922. About ten years later the booster or amplifier and the bone oscillator were added. From this simple apparatus has developed these many transmitters, multiple transmitters, multiple oscillators, multiple bone oscillators and batteries. By multiplying these, several hundred hearing aids are possible, each differing from the other in receiver, amplifier, bone oscillator and battery.

It is apparent that a selection must be made if ears are to be fitted as well as they can be from all those hundreds of possible combinations. The first attempt to do this was with a machine something like the optical trial frame in which the component parts could be placed in such a way that the patient could absolutely hear through the parts that were to compose his hearing aid. The Radio Ear Selexaphone of Pittsburgh was the first analyzer of this kind.

FIG. 1 — (Hayden) — Sonotone Audioscope, a "master hearing aid." Just as the trial frame enables the patient to see through the lens combinations indicated by the retinoscope, the master hearing aid permits hearing through the component hearing aid parts as determined by the audiometer. Three of the four major manufacturers use these instruments: Radio Ear the Selexaphone, Acousticon the Aurogage, and Sonotone the Audioscope. 1. Transmitter. 2. Transmitter adapter replacing the cord of the portable hearing aid. 3. Transmitter receptors, 1, 2 and 3, into which transmitter adapters fit. 4. Transmitter switch connecting receptors 1, 2 and 3. 5. Battery switch. 6. Volt meter. 7. Milliammeter indicating amount of current passing through transmitter. 8. Milliammeter indicating amount of current passing through receiver. 9. Volume control for receiver circuit (taking place of control button on hearing aid transmitter). 10. Volume indicator for receiver circuit. 11. Amplifier base to hold carbon chambers in position for testing. 12. Amplifier carbon chamber, low, standard, medium, high and three specials. 13. Compartments for receivers and cords: A (white) cord, to which the four midget and the original circular air receiver can be attached; —.2 (red), —.4 (amber), —.6 (green) cords to which high, medium or low pitched bone conduction receivers (oscillators) are attached. 14. Compartment for transmitters, air receivers and accessories. 15. Receiver switch, which throws any of the receivers (A, .2, .4, or .6) into circuit. 16. Pilot lights (white, red, amber, green), which indicate which receiver is in circuit. 17. Switch (to the left simplified, to the right amplified) by which the amplifier can be cut either in or out of circuit. The latter (to the left) is essentially Alexander Graham Bell's original hearing aid circuit.

The Sonotone Acoustoscope used by the Author performs the functions of the trial frame and bears the same relation to otology that the trial frame bears to ophthalmology.

This is one of the audiometers that Dr. Newhart has mentioned. Even with the assistance of the master hearing aid that was just shown and the elimination to a large extent of the trial and error method, we had to have something like this for purposes of clinical study as well as for the accurate and rapid fittings of hearing aids, because it is perfectly obvious that the use or trial of several hundred instruments is too fatiguing and time-consuming to use on a patient to advantage.

The audiometer, as Dr. Newhart has said, serves a very useful purpose and one that has not only an octave frequency consisting of eight different tones but has continuous frequency calibrated at the various octave letter steps — such as the Otologist Service Audiometer — is indeed advantageous for detecting the tone islands that Dr. Hartig showed in his picture of the old hearing aid. The receivers or the absorbers of those should be fitted according to an audiogram, as I outlined in a paper in San Francisco at the meeting of the American Medical Association, on hearing aids from Otologists' audiograms.

There are several audiometers similar to this but none, I am sorry to say, is all that most otologists would like to have. None of them meet the requirements of the Council on Physical Therapy of the American Medical Association, but remember that hearing aids are only five years and audiometers only fifteen years old. Perhaps we are expecting to obtain too much perfection right at the present minute.

Even though audiometers do not meet all the requirements, results are fairly consistent. Here is a composite audiogram of twenty-six hard of hearing patients shown for the right ear only (the left ear is similar). You see that the Maco, the Western Electric 6-A, and the Otologist Service are all similar in their outline. For that reason, I do believe that the possession

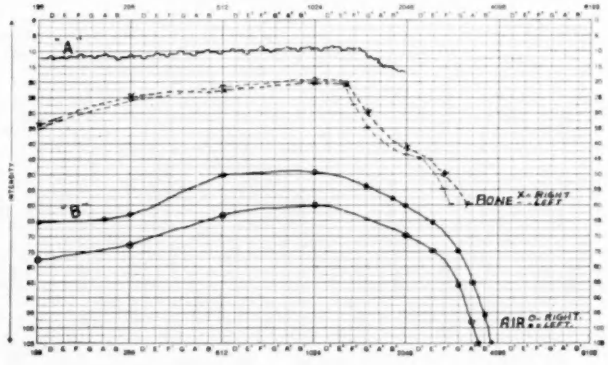

FIG. 2 — (Hayden) — "A" shows the hearing of the patient with and "B" the hearing of the patient without the hearing aid. The difference between the lines "A" and "B," approximately 40 decibels, shows improvement made by the hearing aid. This improvement in terms of distance would be from 2 feet without to 10 feet of hearing with the hearing aid. Audicle prescription: response chart No. B-12, audicle combination C-7-6-4.5 volts, right ear.

of an audiometer at the present minute is the proper view for the Otologist to take, rather than the discussion of which audiometer or whether or not to wait until audiometers are absolutely perfect. He, indeed, and he alone perhaps can contribute more than anyone else to the development of perfection, both in hearing aids and audiometers.

I wish to call your attention to this slide showing the audiogram of a hearing aid. In this case, the air conduction is between 65 and 70 going out at 4,000 and the bone conduction is 35 to 20 going down to about 40 in the upper limit. The hearing can be raised to about a 10 d.b. loss by the use of a properly fitted hearing aid. This audiogram becomes a graphic record of this improvement. Thus, as Dr. Newhart mentioned, it is possible with any of the four instruments — the Radio Ear, the Acousticon, the Sonotone or the Western Electric — to improve hearing from about three feet to about twenty-five feet in favorable cases.

Dr. George M. Coates, Philadelphia: Any paper by Horace Newhart is always well worth listening to and is full of interest to otolaryngologists. Especially is it true of this presentation of a subject which should concern nearly every practicing otologist. Dr. Newhart is one of the few outstanding experts in this country, or any other, thoroughly qualified to speak on all phases of audiometer and hearing aids, the intricacies of which have hitherto been a closed book to the average ear specialist, and we should all be familiar with Dr. Hartig's work on defective hearing. With the great advances made in these instruments during the last few years, it is becoming more and more incumbent on all of us to familiarize ourselves with their workings in order that we may give the utmost in advice to our afflicted deafened and hard of hearing patients. Recent progress seems to indicate that we are at last making a definite approach to the ideal where the otologist may be able to prescribe a custom made hearing aid from his audiometric studies, instead of simply committing his patient to the tender mercies of the commercial producer who has an instrument to sell, and while it is quite possible that we may never, owing to many well-known variables, be able to do this with the precision of the ophthalmologist in prescribing glasses, that is the goal to be striven for. Improvement in audiometers will make diagnosis of hearing defects more accurate, but improvement in manufacturing devices to correct these deficiencies must continue if we are to be able to make the deafened hear as comfortably as those with defective vision can be made to see.

Much has been accomplished in this line in the manufacture of semiportable instruments, but the field for these is limited. Our patients call for a portable apparatus, one too that is inconspicuous, light in weight, economical in current consumption, free from internal and distortion noises, adjustable as to volume, one that amplifies only those frequencies that are most at fault and one that is not uncomfortable to wear. This is a large order which has been successfully met only during recent months by the perfection of truly wearable vacuum tube devices with miniature tubes and crystal receivers. As the miniature electric light bulb revolutionized endoscopic procedures, doubtless in time this problem also will be solved or some other method of equal or better efficiency be evolved.

While at the present moment, the practice of building a hearing aid to meet the individual's requirements is determined by an audiometric study is still in the early stages of experiment, such study is already of the greatest value in enabling the otologist to determine at least the type of aid to be tried out, with air or bone conduction, or whether it is worth bothering with at all. Even this much is a distinct gain, for much unethical advertising, sales talk and ballyhoo in the past has palmed off worthless instruments on desperate people when they could not possibly benefit. Prices charged for instruments must also come in for serious consideration. Audiometers accurate enough to be of value have in the past cost more than many an otologist could comfortably afford—with the present reduction in cost, every otologist will in the near future find that he must be equipped to do this work and courses given here in our Academy, as well as in other societies and schools, are now teaching their uses.

The whole question of hearing aids and their potential importance is just beginning to be understood. As proper glasses increase the efficiency of school children, to a similar degree would individual hearing aids doubtless turn many a supposedly dull and backward pupil into an attentive, bright one and save much of the economic waste of repeated scholastic years.

Contributions such as this one have an important educational value. There should, and doubtless will, be more of them.

Dr. Horace Newhart, in closing: Mr. Chairman, on account of the shortage of time I wish merely to thank those who have so ably carried on the discussion by presenting so clearly features of a subject which we could not dwell upon. This is a subject of growing interest and one which we as servants of the public must be willing to follow up.

Further, I would like to say for the manufacturers that they have rendered a great service in improving their wares and making it possible for so many more people to have serviceable hearing which will enable them to hold their jobs. The use of multiple hearing aids in schools for the deaf is something we should encourage. There would be many more in use if we could get our legislative bodies to make the proper appropriations.

OTITIC SEPSIS DUE TO CAUSES OTHER THAN LATERAL SINUS THROMBOSIS AND PHLEBITIS*

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DURING the course of an otitic infection, a patient may present septic symptoms which may lead one hastily to suspect an infection of the lateral sinus. However, upon careful analysis, one finds that during the course of an otitis media or mastoiditis, pre- or post-operatively, the patient often develops other infections or concomitant complications, such as cervical adenitis, pyelitis, pneumonia or nephritis which present symptoms that are similar to those of lateral sinus thrombosis and phlebitis. It has also been observed that in acute mastoiditis of the hemorrhagic type, sepsis is an early symptom. There is an extension to the general circulation, and the blood stream infection is independent of the lateral sinus.

Post-mastoidectomy, septic symptoms may manifest themselves as a result of: (1) incomplete mastoid surgery; (2) osteomyelitis of the temporal bone; (3) petrosal involvement; (4) extension of infection to other bones of the cranial vault; and (5) contiguous involvement of adjacent soft structures.

All septic conditions present a common group of symptoms such as abnormal temperature, chills, headaches, changes in the blood picture, and occasionally positive blood cultures. The presence of such septic symptoms in conjunction with suppuration of the temporal bone often lead one hastily to ascribe them to lateral sinus phlebitis and thrombosis, rather than to some other pathologic process.

It would not be amiss to discuss briefly the general symptoms of sepsis at this time.

Abnormal temperature is the outstanding symptom of sepsis. The typical temperature is the steeplechase, or the spiked variety. However, the temperature may be sustained during the course of

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an otitic infection and precede the onset of such complications as erysipelas, pneumonia, meningitis, tonsillitis or infection in the opposite ear. Only by watchful waiting and close cooperation, can the true status of the case be determined. Paradoxically the prolonged administration of sulphanilamide while treating a septic patient may produce a temperature of the intermittent type which does not subside until administration of the drug is stopped and it is eliminated from the system.

Chills are usually present in about 50% of septic cases. The infection is often ushered in by a chill. In many instances, where chills are present, the subjective and objective descriptions of the chill may vary from cold extremities to the feeling of chilliness, rigor, and even profound prostrations and cyanosis.

Sweating invariably follows the rise of temperature and should be evaluated when it is present. Profuse sweats will often terminate in prostration.

The presence of convulsions is rather infrequent in adults' cases. They may occur as the result of a toxemia, especially in children's cases.

Cyanosis often supplants the chills as a symptom. It is well to remember that cyanosis is often caused by the administration of sulphanilamide.

The pulse rate invariably parallels the temperature, but in the presence of such complications as meningitis, cerebral or cerebellar abscesses, bradycardia may be present. In prolonged illness with protracted temperature and sepsis, myocardial failure often occurs. The pulse rate then becomes rapid and crosses the temperature on its way down.

At the height of the temperature and during the chill, the patient is likely to be drowsy, apathetic, but as soon as the temperature subsides, the mentality clears and there is a feeling of well being.

The blood culture is positive in 55% to 60% of cases of patients with lateral sinus thrombosis or phlebitis. However, at times we have obtained positive cultures in other complications such as pneumonia, tonsillitis, acute endocarditis, erysipelas, and after operation on the hemorrhagic variety of mastoiditis. This finding in itself should not be the sole basis for diagnosis of lateral sinus thrombosis or phlebitis.

In sepsis, the hemograms will reveal a secondary anemia. A progressive reduction in the hemoglobin and red blood cells in

the presence of sepsis indicates a hemolitic infection. Leukocytosis is present, and a differential study reveals the presence of immature white cells and a shift of the Shilling index to the left.

The spinal fluid does not reveal any chemical or cytologic changes unless there is a complicating meningitis.

We have repeatedly observed a negative Ayer-Tobey in the presence of thrombosis of the lateral sinus and a positive Ayer-Tobey in patients without a thrombus. Therefore, positive or negative findings in the Ayer-Tobey test are only of value in conjunction with the entire clinical picture.

Metastasis occurs in many cases independent of involvement of the lateral sinus. Many infections begin with a septicemia and the ear is not always the primary focus. For example, a sore throat may produce a septicemia and the otitic involvement would only play a minor role in the infection.

We should like at this time to present several selected cases which clearly demonstrate our contention that in the presence of temporal bone suppuration, sepsis may be due to causes other than those of lateral sinus phlebitis and thrombosis.

The cases here presented were carefully studied from all angles, with full cooperation from the internist, the pediatrist, the roentgenologist, and the laboratory.

In the course of our analysis we shall endeavor to emphasize the classical signs and outstanding symptoms as they present themselves. Because of the similarity of symptoms an erroneous provisional diagnosis of sinus phlebitis and thrombosis could readily have been made, but the final outcome of these cases has proven that other forms of sepsis existed.

CASE HISTORIES

Case I

J. B., White, Male, Age 9 years.

Presented the following history: The patient was apparently well until March 27, 1936, when he became ill with a sore throat. Seven days later he developed a right acute otitis media with spontaneous perforation. Three days after this an otitis media developed on the left side and a myringotomy was performed. During the next ten days there was profuse aural discharge, pain over the mastoid process, intermittent temperature ranging from 99° to 104°, headache, chills, recurrent sweats. On physical examination, the heart and lungs were apparently normal. X-ray

examination showed evidence of mastoiditis. Blood count: Red cells and hemoglobin reduced with a marked secondary anemia. White count disclosed a leukocytosis with a shift to the left. Bilateral mastoidectomy was performed, but the sepsis did not subside. On two successive days following mastoidectomy, blood cultures were positive. The organisms isolated were streptococcus hemolyticus. Eye ground examination was negative.

The Ayer-Tobey test was positive on the right side. The mental state of the patient was clear and during the recession of temperature the patient felt well.

A critical analysis of all the symptoms presented pointed towards lateral sinus disease. A revision of both mastoids was performed opening the right lateral sinus, but no pathology was found. The mastoid wounds disclosed some granulation tissue but there was no bony necrosis. The usual therapy was instituted consisting of transfusions (this was prior to sulfanilamide therapy), non-specific sera without any improvement.

One day while examining the patient, petechial spots were noted in the left antecubital region. Shortly afterward the cardiologist obtained positive evidence of the existance of an endocarditis. Under proper therapy the patient eventually recovered.

Comment: This was a case of bilateral mastoiditis, in which the otitic infection either produced a flare-up of a latent endocarditis, or the endocarditis was the result of the otitic infection. In any event, the surgery performed on the lateral sinus did not hasten the patient's recovery.

Case II

J. G., White, Female, Age 8 years.

History: On July 10, 1938, patient went swimming; as a result of this she contracted an upper respiratory infection. Two days later she developed a right otitis media. This was incised 36 hours after its onset. At that time, the temperature was 104.5°, and there was a slight cough and a discharge from the right naris. There was no headache, or pain over the sinuses. For the next ten days the otitis media and sinusitis on the right side persisted. The blood count revealed HGB. 70%, R.B.C. 3,000,000. There was a leukocytosis ranging from 14 to 18,000. The Shilling Index showed a marked shift to the left. Roentgenograms of the mastoid disclosed an exudative mastoiditis, but ostoscopically there was very little evidence of mastoid involvement. Occasionally the

patient complained of chills, and a slight cyanosis was noted. At times there was nausea and vomiting. The blood culture was negative. The temperature was of the intermittent type, ranging between 97°, and 104°. Urine analysis was persistently negative except for the low specific gravity. The outstanding cardinal symptoms were intermittent temperature, chills, sweats, occasional nausea and vomiting, and hemogram changes, in addition to the otitic suppuration and x-ray evidence of mastoid pathology.

Re-x-ray of the mastoid at this time did not reveal any further changes. The condition of the patient became alarming due to the toxemia, the recurrent severe chills which were followed by prostration and the persistent high temperature. In view of the fact that there were no other positive findings to explain this sepsis, the mastoid was explored. At operation, there was very little pathology noted. The symptomatology remained the same, despite the surgery, for ten days. Then there was a sudden drop in temperature, and an amelioration of all other symptoms. The urinalysis at this time disclosed huge quantities of pus and it became evident that the septic symptoms were due to the retention of the pus in the kidney. As soon as the loculation emptied itself, the sepsis subsided.

It is worthwhile to bear in mind that 4% of all abnormal temperatures of unknown origin in children are due to a pyelitis.

Comment: This picture is one of a septic patient with otitic suppuration. There was scant evidence of mastoiditis, but the alarming symptomatology warranted exploration of the mastoid. The true state was eventually revealed as pyelitis.

Case III

C. S., White, Male, Age 3 years.

On March 6, 1935, the child became ill with an upper respiratory infection and bilateral otitis media. Two weeks later upon admission to the hospital, the patient presented the following symptoms: Intermittent elevation of temperature to 103°, nasal discharge, and slight cough. X-ray study revealed infection of the left maxillary and ethmoid sinuses, and a bilateral mastoiditis. The chest examination was negative physically and roentgenologically. Otoscopic findings disclosed profuse suppuration in both ears and narrowing of the canal. There was tenderness over the emissary vein and pain over the mastoid antrum on palpation. The patient appeared toxic and anemic. The hemograms disclosed a leukocytosis ranging between 15 and 18,000, and a Shil-

ling Index to the left. There was a secondary anemia and a tendency towards destruction and degeneration of the leukocytes.

Sixteen days after the onset of the illness a bilateral mastoidectomy was performed. At operation there was found a thickened periosteum, pus, granulation tissue and sequestration. The operation was performed under avertin and local anesthesia. On the first post-operative day the temperature rose precipitantly to 105°, with a slight recession of the temperature for the next 48 hours. On the fourth day post-operatively, the temperature again reached 105°, accompanied with a slight chill and perspiration, and frequent coughing. The blood count now disclosed a marked reduction in the red cells and hemoglobin; the white blood picture revealed a leukocytosis ranging between 14 and 19,000; and a marked shift to the left. Physical examination of the chest was negative. Ayer-Tobey test was positive for involvement of the left lateral sinus. Palpation of the neck disclosed slight tenderness along the anterior border of the sternocleidomastoid muscle. X-ray of the chest at this time was negative.

Six days following the operation the temperature excursions ranged between 97° and 105°. The cough was slightly increased. Another x-ray examination of the mastoid did not disclose any residual diseased bone while x-ray of the sinuses showed improvement. The blood cultures were negative.

Critical analysis of this case disclosed the following outstanding symptoms: (1) Septic temperature; (2) Headache; (3) Slight cough; (4) Positive hemograms; (5) Positive Ayer-Tobey for involvement of the left lateral sinus. This pronounced septic picture in the presence of a bilateral mastoiditis would influence one towards the diagnosis of lateral sinus phlebitis or thrombosis. Chest examination at this time disclosed a slightly prolonged expiration and a few crackling rales. X-ray of the chest was indefinite. In view of the suspicious symptoms referrable to the chest, a conservative attitude was adopted and therefore no surgery was instituted.

Ten days post-operatively the x-ray showed evidence of a broncho-pneumonia. Within 21 days all symptoms subsided, and the patient made an uneventful recovery.

Comment: In this patient there was a bronchopneumonia associated with a bilateral mastoiditis. The septic symptoms after operation simulated lateral sinus phlebitis and thrombosis. It was only the slightest suspicion of the chest that made us adopt a

conservative attitude and thus avoid an unnecessary operation of the venous structure.

Case IV

W. B., White, Male, Age 6 years.

The patient was ill with an upper respiratory infection in April of 1938; after 3 days the child developed severe pain in the left ear with a temperature of 103°. A myringotomy was performed by the family physician, this was followed by a serosanguineous discharge without abatement of the symptoms. Five days after the onset of the illness, the right ear became involved. Otologic examination at this time disclosed an otitis bullosa externa. The left ear also presented blebs in addition to a serosanguineous discharge. The blebs were not punctured but were treated medically. On the seventh day there was a sudden rise in temperature, with chills. Otoscopic examination now disclosed a bilateral otitis media which upon myringotomy released thick purulent discharge from both ears.

Nasal examination disclosed a bilateral suppurative maxillary sinusitis. Subjectively the patient complained of a frontal headache, heaviness of the eyes, occasional nausea and vomiting and retro-orbital pain. The sinuses and the ears were treated, but the temperature, sweats and prostration continued. Temperature ranged between 99° and 103.5° for 10 days. The blood counts disclosed red blood cells 3,200,000 H.G.B.; 70% white count; leukocytosis 15,000 to 23,000. The otorrhea was profuse and had a greenish tinge. Otoscopically and clinically there was evidence of bilateral surgical mastoiditis. Roentgen findings disclosed antra and ethmoid pathology and revealed active destructive mastoiditis.

On the 19th day after the onset of the illness a bilateral mastoidectomy was performed. The findings at operation were necrosis of bone with cavitation. For 4 days post-operatively there was a subsidence of the temperature to 100° after which there was a recrudescence of the septic symptoms. There were also several mild chills. All sutures were removed, the wounds were opened widely and inspected and no residual pathology was found in the mastoid region.

Ayer-Tobey test was positive on the right side. Blood culture was negative while the hemograms continued to show evidence of infection. Therapy consisted of blood transfusions, sulfanilamide was administered by mouth and prontysil was administered hypodermically. Antrum puncture disclosed thick pus on the right

side. We were unable to ventilate the left maxillary antrum because of the obstruction of the osteum. Irrigation was accomplished with the use of two needles through the inferior meatal route. We were unable to catheterize the natural ostia at any time.

The septic symptoms continued for three weeks without improvement until the infection in both antra cleared up.

Comment: These septic symptoms were due to infection in the nasal accessory sinuses. We recently had a case of sphenoiditis that presented the identical clinical picture.

Case V

Patient: K. J., White, Male, Age 9 years.

Three weeks prior to admission the patient had influenza. This became complicated by a right suppurative otitis media. Myringotomy was performed without abatement of the symptoms. One week later a simple mastoidectomy was performed by another otologist.

Upon admission to the Temple University Hospital the following symptoms were present: there was a temperature of 105°, slight nuchal rigidity, profuse discharge from the middle ear and the mastoid wound. There was a secondary anemia present, with leukocytosis of 19,000, and a Shilling Index to the left. The child complained of headache, with fleeting labyrinthine symptoms such as vertigo, and inconstant nystagmus. The Crowe-Beck sign was positive. There was evidence of increased intracranial pressure, as evidenced by the increased spinal fluid pressure and the choked disc of two diopters. The Ayer-Tobey test was positive.

The post-auricular wound was slightly discolored, with a profuse and offensive purulent discharge. The Greisinger sign was present. Palpation of the neck disclosed pain and tenderness along the anterior border of the sternocleidomastoid muscle. The blood culture was negative. The roentgenogram revealed residual pathology of the mastoid process.

Revision of the mastoid disclosed that an incomplete mastoidectomy had been done and many necrotic cells were found in the perilabyrinthine and zygomatic regions.

Comment: This case presented symptoms of sinus thrombosis and phlebitis and was referred to us with a provisional diagnosis of lateral sinus involvement. After complete mastoidectomy, all signs of sepsis disappeared and the patient made an uneventful recovery.

HEMORRHAGIC MASTOIDITIS

Acute hemorrhagic mastoiditis is a distinct clinical and pathological entity. From the onset of the acute otitis media, septic symptoms are often present and are ushered in with chills, high temperature, marked prostration, extreme restlessness and an overwhelming toxemia.

In infants and children characteristic symptoms are extreme illness, gastrointestinal disturbances, high temperature and meningeal symptoms.

The otitic picture begins with a severe diffused redness of the tympanic membrane without any marked bulging.

In hemorrhagic mastoiditis there are two distinct classical pathognomic findings when myringotomy is performed: (a) myringotomy reveals the escape of a small amount of thin serosanguineous fluid. The discharge remains scanty and is of a serosanguineous consistency throughout the course of the infection; (b) there is profuse bleeding following myringotomy which is often difficult to control.

The pain and the sepsis persist despite the fact that myringotomy has been performed. The patient is so ill and septic during the period of otitis media that one is likely to become unduly alarmed and hasten to perform a simple mastoidectomy.

When mastoidectomy is performed early the septic symptoms become more pronounced. At this time the laboratory findings may disclose a positive blood culture, and the most prevalent organism found is the sterptococcus hemolyticus, although occasionally one finds the bacillus influenza.

The blood count shows a secondary anemia with a diminution in the hemoglobin and red blood cells. There is a leukocytosis, ranging from 15,000 to 20,000; a polymorphonuclosis, ranging from 80% to 90% with a marked Shilling Index shift to the left.

Occasionally in hemorrhagic mastoiditis we find venous congestion resulting in the engorgement of the scalp veins, marked edema over the mastoid and the appearance of the so-called Greisinger's sign. The eye grounds may show a choking of several diopters.

Comment: Sepsis, prostration, chills, and occasional positive blood culture, positive hemograms, and occasional choked disc would lead one to suspect lateral sinus phlebitis or thrombosis

but in actuality, these symptoms are due to a hemorrhagic mastoiditis.

OSTEOMYELITIC PHENOMENA

There are two types of osteomyelitic phenomena, viz.: The sub-acute and the fulminating. The sub-acute may present septic phenomena but in a very short time it can be readily diagnosed.

We are primarily interested in the acute fulminating type which may involve the mastoid process, the petrosa or any portion of the temporal bone.

The acute fulminating type of osteomyelitis is followed by a train of symptoms, the most frequent being an elevated temperature which at times is sustained, remittent or intermittent, accompanied by occasional chills, sweats and prostration. We might stress the significance of the severity of the symptoms and rapidity of the deterioration of the general condition of the patient. As a general rule, the course is a stormy one due to the fact that the infection spreads by means of continuity and contiguity. This type of case may sometimes be confused with a lateral sinus phlebitis or thrombosis.

There is a form of acute osteomyelitis of the petrous portion of the temporal bone, described by Ramadier, and most frequently found in children, which spreads through the hematogenous route. It occurs in the young, and produces kidney and lung infarcts; positive blood cultures may be obtained and the general symptomatology is most severe. This type of infection causes wide destruction and has no respect for anatomic boundaries. Fortunately, this type of osteomyelitis is infrequent. These symptoms run parallel with those of lateral sinus phlebitis and thrombosis. The sepsis here is due to the osteomyelitis and surgery of the lateral sinus is contra-indicated because the lateral sinus does not participate in this type of sepsis.

Comment: When early mastoid surgery is performed before localization takes place, the patient faces a rather stormy course with a protracted convalescence. During this period the septic symptoms closely simulate those of sinus phlebitis or thrombosis.

In our experience we have found that there is no emergency in the institution of surgery in lateral sinus phlebitis and thrombosis; by conservative measures time may reveal that the sepsis was due to other causes than that of lateral sinus phlebitis and thrombosis.

SUMMARY

1. Each case of sepsis is entitled to deliberate and deep consideration from a medical and surgical standpoint.

2. The elements concerned in the causation and manifestation are so numerous that it becomes easy to err. Therefore, the surgery of the lateral sinus should not be an emergency measure but should only be instituted after a period of conservative treatment and long deliberation.

3. The pathology, the anatomic variations, the virulence of the organism, and the resistance of the patient are factors which must be considered.

4. Such infections as erysipelas, lobar pneumonia, endocarditis, empyema, and tonsillitis may be primary, concomittant with or sequella to temporal bone suppuration and may produce virulent septic symptoms which can readily be misinterpreted as lateral sinus phlebitis and thrombosis.

DISCUSSION

Dr. Claude C. Cody, Jr., Houston, Texas: Dr. Ersner has presented and proven two propositions in his paper this afternoon. The first is that the sepsis accompanying acute suppurative otitis media and acute mastoiditis is due to causes other than thrombosis of the lateral sinus; and second, that thrombophlebitis of the lateral sinus is not necessarily an emergency surgical condition. As a corollary to these it follows that sufficient time can be taken, as Dr. Ersner has well said, to make a differential diagnosis between the various causes of sepsis associated with aural infection. Fever is the cardinal symptom of sepsis; and the temperature occurs in three types, the steeple, the pneumonic and the typhoidal. In thrombosis of the lateral sinus, the steeple variety occurs six times as often as the other two combined; but in acute purulent otitis media, the pneumonic is far more frequent.

To the causes of sepsis concomitant with otic infection mentioned by Dr. Ersner, we on the Gulf Coast in Texas, are accustomed to add two others, malaria and brucellosis. Malaria and brucellosis associated with aural infection are rare, but with us must be eliminated in the differential diagnosis. Also, malaria and thrombosis of the lateral sinus may be present at the same time. This occurred in a patient of one of my associates, Dr. Lyle J. Logue, who demonstrated at operation a thrombus in the lateral sinus and plasmodium malariae in the blood. Incidentally, the dosage of quinin sufficient to cure the malaria had no effect on the septic temperature. The key to the differential diagnosis of acute brucellosis is the undulant temperature and a positive agglutination in dilution above 1:200.

The differential diagnosis between thrombo-phlebitis and pneumonia in infants and small children can occasionally be made only by an x-ray

examination. The methods of physical diagnosis even in the most skillful hands so frequently fail in these cases, that the chief reliance in the differentiation must be placed on the radiogram.

A series of blood counts made at regular intervals is valuable. It has been my experience that a shift of the Schilling hemogram to the left suggests an uncovered mastoid cell; while a falling red blood cell count and hemoglobin percentage with increasing leucocytosis and a shift of the Schilling hemogram to the right is regularly associated with a thrombosis of the lateral sinus. There is so much difference in opinion on the diagnostic value of the blood culture that it seems obvious the reports are not based on the same technic. While the technic of the blood culture is not essentially an otological problem, the interpretation of its results in the differential diagnosis certainly is. If, after investigation, one technic showed superior accuracy, its endorsement by the Academy would facilitate its introduction in our hospital laboratories more than the unsupported request of an attending otologist.

Dr. Ralph A. Fenton, Portland, Ore.: This paper calls together for review a number of conditions, in themselves well known, which one is glad to find associated together for our aid in the differential diagnosis of otitic sepsis.

In addition to the items mentioned by Dr. Ersner, one might point out the occasional violence of symptoms disclosed by the presence of acetone and diacetic acid in the urine, so-called acidosis. That may turn everything upside down, as we all know, and make us think of a lot of very serious complications which do not at all exist and may easily be done away with by very simple means.

One should also recall the bizarre symptoms and the septic signs coming from exacerbations of chronic sphenoidal suppuration — that is particularly hard to pick up in a young child — dizziness, central headache and continuous fever. This was mentioned but not stressed in Dr. Ersner's excellent presentation.

After all, individuals suffering from otitic sepsis must constantly be regarded as vulnerable in many regions. The otologist must never forget that such patients are sick all over until their resistance has been brought back to normal again.

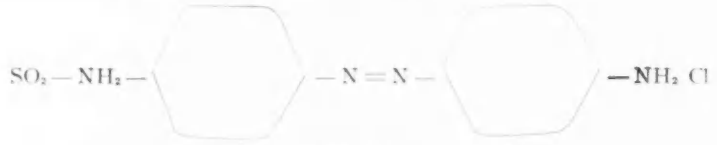
Dr. Matthew S. Ersner, in closing: I came up here again merely to thank the discussors. I really haven't very much to add, because there are so many conditions that will produce sepsis. The only reason we presented this paper was due to the fact that only two per cent of all these cases in our experience have otitic complications or present signs of thrombosis: but, in 90 per cent of these cases we are tempted to think of the lateral sinus. What happens in our service, I am sure, happens in others. I believe that before we attempt to obliterate the lateral sinus, or to open it, we should remember just one little figure, that only two per cent produces signs of phlebitis or thrombosis, and should therefore think of other causes before one attempts to do anything to the venous structures.

USE OF SULPHANILAMIDE IN OTOLARYNGOLOGY

KARL MUSSER HOUSER, M.D.
PHILADELPHIA

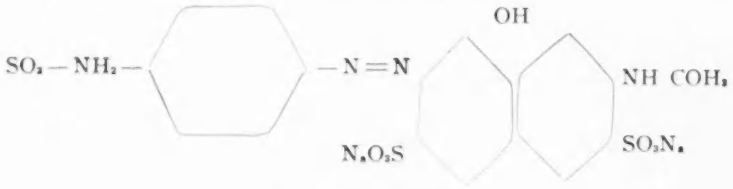
THE HEMOLYTIC streptococcus occurs so frequently in infections of the ear, nose and throat that the Otolaryngologist has a particular interest in any remedy that has promise of combatting such infections. Since the original paper of Domagk[1] in February, 1935, dealing with a new dye stuff developed by Mietzsch and Flarar which, in vivo, was antagonistic to the streptococcus. Medical literature has been flooded with papers dealing with this and similar compounds. Naturally there is considerable confusion as to the indications, contraindications, methods of action and limitations of these drugs, and one writing on the subject today can do little more than attempt to systematize and catalogue the experiences and ideas of those who have already worked with them.

The original compound which received the trade name of Streptozon, and later Prontosil, was chemically Sulfamido diamino azo benzene with the following formula:



PRONTOSIL

This chloride, although slightly soluble in water was modified in order to secure a more soluble form that could be used effectively for injection therapy, and finally Prontosil Soluble was obtained, having the chemical formula:



PRONTOSIL SOLUABLE

These compounds were found to have almost identical effects on streptococcic infections. On first glance their chemical structures seem to be quite different, but it will be noted that the sulphonamide radical is present in each and in the paraposition to the azo group. This was soon discovered and at once a number of investigators (2 a and b) found that the para-amino-benzene —sulfonamide radical was really the important one, and the simpler compound today known as sulphanilamide was developed:

SULPHANILAMIDE

Here we have the amide and sulfonamide radicals in the paraposition of the benzene ring. Many synonyms and trade names for sulphanilamide have arisen which add to present existing confusion of nomenclature. Some of these synonyms are 1162 F, streptocide, sulfamidyl, prontosil album, prontylin, para amino benzene sulfonamide, sulfonamide-P, antistreptin, septoplix and supron.

One need only read a few of the noteworthy results secured with these drugs to be convinced that our present problem is not to decide if these drugs are worth using but to know the best methods, dangers, indications and contraindications of their use.

A large amount of well controlled laboratory work has proven conclusively that sulphanilamide can, in most instances, control or cure infections with the hemolytic streptococcus in laboratory animals. Certain unexplained phenomena do, however, occur. Some strains of the hemolytic streptococcus seem to be affected little if at all by the drug. Then again some laboratory animals, although apparently cured, will die some weeks after cessation of treatment and postmortem demonstration of the streptococcus in the blood and other organs is possible.

The exact mode of action of this drug is not yet determined. No one has been able to demonstrate a direct antibacterial property. In vitro the drugs have little if any effect. A tremendous amount of experimental work has been done, but as yet I believe we are safe in saying that no one knows exactly how infections are controlled or cured by this type of therapy. About as far as

we can go is to say that something happens permitting the body defenses to overcome the infection.

These drugs have been administered by mouth, intravenously, subcutaneously, intramuscularly and intrathecally. The fundamental problem is the maintenance of an effective blood concentration. More recent writers maintain that this can be secured without difficulty by oral administration, provided the drug can be introduced into and retained by the stomach. Opinions differ somewhat upon the optimum blood concentration. The recent work of Osgood[3] shows the effectiveness of relatively low blood values. On the other hand Long[4] who has had extensive clinical experience advocates a minimum concentration of 10 mg. per 100 cc. of blood for severe cases. For a present working basis it is probably sensible to secure a concentration of 5-10 mg. per 100 cc. for the milder cases, reserving more heroic doses for the more fulminant infections. Such concentrations can usually be secured and maintained in adults by using daily divided doses of 4-5 grams. In children with severe infections 1.0 gm. for twenty pounds of body weight has been recommended by Schwentker, Clayson, Morgan and Lindsay,[5] the total dose for twenty-four hours, however, not to exceed 5 gms.

In spite of any set rules each case is a law unto itself and must be so regarded. Undoubtedly some individuals are more sensitive to the drug than others. Therefore it follows that determination of blood concentration of sulphanilamide should be a part of the technique of administration in all severe cases when high doses are given. In addition blood counts at least every forty-eight hours should be available in order to detect any signs of dangerous leukopenia or anæmia. If such checks are available needed increases or decreases in the amount of drug administered can be more intelligently estimated. This means that cases so treated must be hospitalized, unless such laboratory facilities are available in the home. In addition, no patient should receive this drug unless he is constantly under the care of a physician during the period of administration.

If we dismiss the fatalities that followed the use of elixir of sulphanilamide as irrelevant, which we are justified in doing since it has been conclusively shown that the solvent and not the drug was here to blame, we find a rather long list of complications that have followed the use of sulphanilamide. Although formidable in appearance, close analysis shows relatively few really

serious conditions to have arisen following its use; enough, however, to justify extreme caution. A fairly complete list of the untoward reactions described to date would read as follows:[6]

UNTOWARD REACTIONS OF SULPHANILAMIDE

1. General: Malaise, faintness, vertigo, headache, tinnitus and lassitude. (Common but relatively trivial.)
2. Central Nervous Symptoms: Mental confusion and optic neuritis. (Rare and apparently not producing permanent damage if recognized in time.)
3. Gastro-intestinal: Nausea, vomiting, pyrosis, anorexia, abdominal pain. (Common and sometimes distressing. Not serious.)
4. Hepatic: Acute hepatitis with jaundice and cholemia. (Rare but potentially serious.)
5. Renal: Transient hematuria and albuminuria. (Rare and trivial. If combined with hepatitis may be serious.)
6. Cardiac and Circulatory: Shock, precordial pain, anoxemia and fresh cardiac damage in predamaged hearts. (Rare and thus far not serious.)
7. Cyanosis: Prompt or delayed. May be progressive. (Usually mild and common. Not serious if not due to sulphemoglobinemia.)
8. Acidosis: Not of clinical importance.
9. Cutaneous: Morbiliform, scarlatiniform, urticarial, vesicular and purpuric lesions. (Fairly common. Not serious.)
10. Febrile: Irregular. Usually pyrexia after week of treatment. (Not common. Not serious.)
11. Anemia: Acute progressive hemalytic anemia. (Rare but serious.)
12. Leukepenia: Delayed but rapidly developing agranulocytosis. (Rare and serious.)

Thus we see that most of the reactions noted to date are not of necessity serious, and most of the severe ones can be forestalled if the physician is on guard and withdraws the drug in time. The drug should never be given along with sulphates, as the combination tends toward the production of sulphemoglobinemia. Coal tar derivatives also should be withheld. Some advocate the administration of bicarbonate of soda with the sulphanilamide.

In Otolaryngology we have a number of conditions where the decision as to the use of sulphanilamide must be made. We know that an acute cold is caused by a virus and that no one has been able to demonstrate that sulphanilamide is effective against such conditions. We know too that the virus stage of the cold is of short duration and that the streptococcus and pneumococcus are common secondary invaders. Should we, therefore, use moderate doses of sulphanilamide during the virus stage, in an attempt to prevent the development of the secondary stage and the complications that so frequently occur during this period?

We know that most middle ear infections are caused by the hemolytic streptococcus or pneumococcus. Is it right, therefore, to treat all cases of otitis media with sulphanilamide? There is much laboratory evidence that demonstrates such therapy does help in preventing the spread of a local infection. In acute mastoiditis, after central necrosis has occurred it is impossible to greatly influence the abscess already formed because the blood containing the drug no longer reaches this region. In spite of this it should be given as a prophylactic measure against the spread of infection to the blood stream or cranial cavity.

The results of the use of the drug are so striking in streptococcic meningitis and favorable although less striking in pneumococcic infections of the meninges, that we probably all will agree that it should be used in adequate dosage where meningitis exists or threatens. Many will not approve of its use at the onset of acute sore throats and colds. The proper evaluation of this question will only be settled after large series of treated and untreated control cases have been studied, and the incidence of complications appearing in the treated cases compared with the untreated.

Some years ago we held blood transfusion in reserve for cases in extremis. This is no longer so, and this form of treatment probably finds its widest use as a supportive measure for the purpose of preventing the development of the more serious conditions. Possibly we should feel somewhat the same about the administration of sulphanilamide and not withold its use until serious complications have developed. If given early in acute otitis media, acute streptococcic throats, pneumococcic and streptococcic sinus conditions, should it not, if effective, prevent the development of the graver complications? Again, the answer to this question will not be forthcoming until statistics from many series of treated and untreated control cases are available.

SUMMARY

1. In Sulphanilamide the Otolaryngologist has a valuable weapon against infections caused by the hemolytic streptococcus, and in a lesser degree, the pneumococcus.
2. Only cases under constant observation of the physician should receive the drug.
3. The physician should have adequate knowledge of the proper method of using the drug and of its possible complications.
4. When a large dosage is indicated occasional blood concentration estimations should be made.
5. Frequent blood counts are necessary to detect any indications of leukopenia or anemia.
6. Most cases receiving adequate dosage will show some degree of cyanosis.
7. The serious toxic effects of the drug apparently are the result of idiosyncrasy.

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DISCUSSION

DR. HENRY M. GOODYEAR, Cincinnati: The advent of sulfanilamide has added a new hope and proven a life saving measure in many infections formerly considered beyond medical or surgical aid. Three years ago we did not expect recoveries in pneumococcus and streptococcus meningitis. Today with the use of sulfanilamide, and frequent drainage of the spinal fluid, these cases are recovering in astounding numbers.

At the Children's Hospital in Cincinnati we have had a recovery of more than 50 per cent in streptococcus meningitis since the advent of sulfanilamide. Up to 1937 all our cases of pneumococcus meningitis had died. In 1937, however, with our first trials with sulfanilamide in four cases of pneumococcus meningitis, we had two recoveries, one of Group III and one of Group V.

When the patient is able to swallow, I think sulfanilamide should be given by mouth with an equal amount of soda. I can see no advantage in

giving it in the vein, subcutaneously, or intraspinally, if it can be retained per orum. For, like urea, it is rapidly and evenly distributed throughout all tissues of the body except bone. Giving it intravenously tends to increase the fluid in the vessels with an increased elimination of the drug through the kidneys, thus defeating its own purpose.

Normally, spinal fluid contains 98 per cent water while blood plasma contains 92-93 per cent water; this small variation probably accounts for the slight variation in the concentration of sulfanilamide in the blood and spinal fluid. The concentration being practically identical in the blood and spinal fluid if the water volume of each is considered. Thus I can see no indication for intraspinal administration. Diffusion takes place so rapidly that it would be almost impossible to introduce sufficient of the drug intraspinally to produce a concentration of any medicinal value.

If the patient is comatose sulfanilamide should be given subcutaneously, introducing a one per cent solution in normal saline or, preferably, one per cent in Hartmann's alkaline solution, which eliminates the worry of a possible acidosis. To a patient weighing 130 to 160 pounds 600 cc. is given as the initial dose, and 400 cc. in six hours and then repeated twice a day for four days, with a check on the concentration in the blood.

Prontosil may be less disturbing to the stomach, but it requires two and one-half times as much to raise the blood level as compared with sulfanilamide, for its efficacy depends on the sulfanilamide liberated.

Sulfanilamide acts best in Beta haemolytic streptococci infections. To date, six different types of streptococci have been identified, and Rebecca Lancefield of the Rockefeller Hospital has recently devised a method, whereby they may be identified within thirty minutes, whereas, it usually requires four or five days.

Significant is the fact that 70 per cent of all streptococci infections in humans are Beta haemolytic. Thus in acute otitis media and early spontaneous rupture where haemlytic streptococci are present in 90 per cent of the cases, a trial of sulfanilamide for three or four days seems justifiable.

It has been my practice to reserve the use of sulfanilamide for the unusual and often acute fulminating types of infection. Recently, in a baby of ten months an erysipelas cleared within 48 hours, and in a man age 38, with erysipelas of the external ear and a temperature of 104, showed a normal temperature 24 hours after the administration of sulfanilamide.

Recently a boy age 8 came to my attention, who had had intermittent temperature for two weeks, and the only physical finding was moderately enlarged glands of the neck. The temperature was 105 and the child was flighty and semi-delirious. With sulfanilamide the temperature was normal in 24 hours, and remained so with a rapid recovery.

Six weeks ago I saw a woman age 45 with an acute mastoid of three weeks duration. There was mastoid tenderness, a swelling of the posterior canal wall. The patient looked very ill and for ten days large doses of codeine and aspirin had been necessary for headache and severe occipital pain. A simple mastoidectomy seemed clearly indicated. Ninety grains of sulfanilamide were given in 24 hours. The pain was relieved immediately; the aural discharge ceased on the third day, and the patient returned to her home in one week without operation or further trouble.

In my cases sulfanilamide, when effective, has acted favorably in 1 to 4 days, and now where no improvement occurs in 4 days the drug is discontinued.

While sulfanilamide is now being hailed as a boom to therapeutics, tomorrow it may be passe. G. M. Evans and F. Garford, London, recently reported astounding results with a product called 2-sulfanilamide pyridine, which is ten times as effective as sulfanilamide, and one-tenth as toxic, thus it should be 100 times better than sulfanilamide.

DR. FLETCHER D. WOODWARD, University, Va.: I would like to commend Dr. Houser on the timeliness of his subject and on the way in which he has presented it. There is certainly no question as to the value of sulfanilamide for we have all seen it perform "miracles." Its introduction marks another milestone in the progress of Medicine, comparable to the introduction of Insulin, liver therapy, and the many other discoveries of the past which we have regarded in the same way.

However, because of the relatively short time that it has been in clinical use, there is still considerable confusion as to the indication, contraindication, methods of action, and limitation of its use, as Dr. Houser says, and until sufficient time has passed, we can only make progress toward the solution of these problems by each individual setting down his own personal experiences with the drug. With this end in view, I wish to briefly give my conclusions to date in regard to its use, in our experience, at the University of Virginia Hospital.

Administration in the great majority of cases has been by mouth, using the tablet in conjunction with bicarbonate of soda, and for the first 48 hours giving approximately 1 grain to 2 lbs. of body weight each 24 hours, the subsequent dose being determined by the condition of the patient at that time. This table of dosage has been sufficient to maintain the blood concentration at from 5 to 10 mg. per 100 cc.

It has also been our custom to maintain a careful check on the blood, and although there have been many cases in which the drug was stopped, we have not had any serious complications or deaths.

I quite agree with Dr. Houser's discussion on untoward complications, and see no reason why we should have more than an occasional serious complication, when these early signs are carefully watched for, except perhaps when one is allergic to the drug. However, if the drug is prescribed and used widely by the laity without proper medical supervision, there will no doubt be many serious effects from its use. These, however, in no way condemn the drug.

It has been our custom to prescribe it practically in all acute middle ear and mastoid infections, and as a result the private practice of otology has been the only thing to suffer a severe complication, a financial one; for we have had to perform only one simple mastoidectomy in the past year, as you will see from the accompanying table. This result has not been so dramatic in the clinic because of the lack of proper home care and because so many are brought in with well developed mastoiditis, which as always necessitates operation.

It is a simple rule to remember: that the drug serves to prevent and treat mastoiditis, but the indications for operation are still the same. It serves to prevent and treat complications, but the operative indications in these are also still unaltered.

Under the dosage mentioned above, we have been happy to observe many cases of meningitis recover; and many cases of septicemia do likewise with a shortened period of convalescence. Among these were two dramatic recoveries, one with Type III pneumococcus meningitis from a middle ear infection, and one of an extradural abscess from a ruptured petrous apex cell, due to the hemolytic streptococcus with an associated cavernous sinus thrombosis and paralysis of the third, fifth, sixth, and seventh cranial nerves.

In infections of the sinuses and upper respiratory tract, the results have not been so dramatic as in those of the ear, probably because so many secondary bacterial invaders are present. At this point, I would like to mention our disappointment in it, in staphylococcic septicemia and osteomyelitis of the frontal bone. We have recently lost two patients with an acute osteomyelitis of the frontal sinuses and bone and early subdural abscesses. They were adequately treated with all means at our disposal, including sulfanilamide, but the course was rapidly fatal. The same unhappy result was also experienced in two cases of cavernous sinus thrombosis and septicemia, yet in a third case of nasal furunculosis and staphylococcic septicemia a recovery was obtained; but we did not feel that sulfanilamide could be definitely credited, since it was only one of many measures used. A cavernous sinus thrombosis was strongly suspected here, but could not be proved. In pneumococcic and streptococcic meningitis, however, we can again cite several dramatic recoveries.

We use sulfanilamide in all acute sinus and throat infections, and feel that it probably influences the course; but since many other secondary invaders are present, one should not expect too much. The same holds true in chronic infections, and for that reason we have used it very little in chronic cases.

In acute infections in the lower respiratory tract, it has again exerted a most favorable effect in many instances, especially in those severe laryngotracheo-bronchial infections in children, and in the pneumonias when due to organisms susceptible to the drug.

Dr. Dupont Guerry, in his experimental work on rabbits done at the University of Virginia, concluded that sulfanilamide exerts a bacteriostatic action on hemolytic streptococci in vivo, that there was no evidence of bactericidal action, that the granulocytes were most important in the control of infection, and that the monocytes come in later to reinforce the granulocytes in the destruction of the organism. He was unable to evaluate the other components of the reticulo-endothelial system in this defense mechanism.

In conclusion: we have a simple and comparatively safe remedy for the treatment of the heretofore most dreaded of infections, and we should honor the discoverers and early proponents of sulfanilamide for giving us such an effective weapon in combatting them.

I have one slide which I should like to show. This slide is merely sketched covering a ten-year period. The broken line represents the total

number of simple mastoidectomies performed in the hospitals in Charlottesville, Virginia, and the solid line represents the total number of simple mastoidectomies performed on private patients alone. You will notice beginning in the year 1928 and going on through to the eight months of 1938, we have reduced this total number of cases. Whether this means anything or not, no one knows, except that it does give evidence that in the past year there has been only one simple mastoidectomy performed on a private patient. That has never happened before. The total number of cases is undergoing a sharp decline. We hope that sulfanilamide is responsible for this, and I think if this record is continued over a period of a few more years, you will have evidence whether it has something to do with it or whether this is just a coincidence.

DR. SAM E. ROBERTS, Kansas City, Mo.: The Transactions next year are going to have added value to all of us after Dr. Woodward's statement. When the income tax collector comes around, you can refer to his discussion of this paper and explain why your tax is less.

My written discussion consisted of four words: fear, proceed cautiously, oxyhemoglobin, and enthusiasm.

Since there has been so much in the lay press and the medical press about the dangers of sulfanilamide — of course the lay press hear only the elixir side of it — the word is indelibly imprinted in their mind and when you write a prescription for sulfanilamide they will scrutinize it very carefully and say, "Isn't that a dangerous drug?" So I have proceeded by writing the prescription with the trade name.

That fear isn't altogether limited to the layman. There is a fear among the profession. In our specialty we do most of this prescribing. I do not believe that the other specialties in medicine, except perhaps the urologists, use it anywhere near as frequently as we do. Hence we have the opportunity of having the greatest experience with it.

I have been using it since March, 1936. The first case, as often happens, is the most dramatic. This was a case of meningitis diffuse. A child, unconscious, was brought into the hospital with all the cardinal signs of meningitis which usually has a fatal termination. She had been unconscious three days; was dehydrated. We did a very extensive mastoidectomy, including both the superior and posterior fossæ and the lateral sinus, and administered sulfanilamide. Much to our surprise, in four days the child was seemingly on the road to recovery and went on and recovered completely.

The next case was a case of meningitis following a suppurative labyrinth which did not recover. Since that time the percentage has been approximately 50 to 60 per cent of recoveries. I have used it in sinus thrombosis, and I do think one point that has not been stressed perhaps as much as it should be is the value of multiple small transfusions in meningitis or in sinus thrombosis. We cannot give up all of our past therapeutic means even though we have a valuable addition to our armamentarium.

The question of oxyhemoglobin was mentioned. About a year ago in St. Louis I heard Hartman discuss informally the value of giving methylene blue along with sulfanilamide. If he has published anything on the subject, it has escaped my knowledge. I have not seen it. He advised the use of one grain of methylene blue with each dose of sulfanilamide. Methylene blue forms hemoglobin, the same as sulfanilamide, but he brought out the fact that the two working together lessened very much the advance of oxyhemoglobin.

As Dr. Houser brought out, most of the complications are inconsequential. Though it has always been an alarming symptom, I think we have attached too much significance to the fact that the patient becomes somewhat cyanosed. In the beginning I reduced my dosage markedly when the patient became cyanosed. Now if the case is serious I go ahead with its administration even though the patient does show a mild cyanosis.

As for the question of enthusiasm, in our solved problems we should not adopt radical changes in our therapy, but in our unsolved problems, such as meningitis and the septic sore throats, which take off quite a number of people each year, we are justified in being a little more heroic, and when we have handed to us a reasonably safe drug, such as we have with sulfanilamide, we should become enthusiastic about it.

My rule in office practice, with ambulatory patients, is to give them a prescription for not to exceed thirty tablets, marking the prescription, "Do not refill." In that way, I know that the patient isn't going to continue too long, and isn't going to take too great a dosage of it. We may know five years from now that this has after-effects of which we are not conscious today, so we should proceed cautiously, but nevertheless our enthusiasm should be great for this drug.

I want to thank Dr. Houser for presenting it in a most concise way.

THE TECHNIC OF LARYNGOFISSURE FOR CANCER OF THE LARYNX*

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LARYNGOFISSURE, laryngotomy, thyrofissure, thyrotomy — all of these terms indicate a division of the thyroid cartilage in the midline. They do not include excision of the diseased tissue within the larynx. The operation might more properly be designated: Larynogofissure with Excision, so as to convey a better understanding of the nature and extent of the procedure.

The origin of laryngofissure is usually traced to Desault,[6] who first suggested it, some time during the latter part of the eighteenth century. In those days the operation was termed Laryngotomy. Laryngotomy, or opening of the larynx, was considered a subdivision of Bronchotomy. The other subdivision was Tracheotomy. Laryngotomy consisted of dividing the cricothyroid membrane transversely or of cutting the thyroid cartilage longitudinally. In 1812 Bichat[1] wrote that laryngotomy "was not described except by some authors who restrained the use of it too much and retrenched the process." Bichat preferred laryngotomy to tracheotomy. He claimed for it greater accessibility, less hemorrhage and a simpler technic.

A brief account of the incident which led Desault to suggest laryngotomy might be of interest:

Desault (1744-1795) was called in to see a man who had aspirated a cherry. It was determined that the cherry had lodged in the left side of his larynx. Desault advised division of the thyroid cartilage, but the other consultants rejected the operation because of "the danger of hemorrhage, the difficulty of dividing the thyroid cartilage, and of the possibility of an aerial fistula after operation." When the patient died, two years later, autopsy revealed that the cherry in his larynx had caused his death.

Since the time of Desault the operation has passed through many phases. In the early days, surgery was performed without

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accurate diagnosis, without anesthesia, with crude instruments and with awkward postoperative care. As a result there were many serious sequellae and failures.

Brauers[2] has the credit for having performed the first laryngofissure, in 1833. Without anesthesia and without a tracheotomy he exposed a papillomatous growth of the larynx and cauterized it. This was an unusual achievement. Buck[3] performed the first laryngofissure in America, in 1851, while Gibbs[9] is credited with having introduced laryngofissure in England in 1864. Gibbs remarked that the operation ought to be one of the simplest performed upon the neck.

The introduction of the laryngoscope by Manuel Garcia[8] of London, in 1855, resulted in an increased interest in laryngeal disease, with a consequent increase in its surgery. During the following fifty years the operation was performed by many distinguished laryngologists. Laryngofissure has had its greatest vogue in England. Gibbs,[9] Durham,[7] Butlin[4] and Semon[14] did much to develop laryngofissure. Since 1900 Tilley,[16] Lack,[11] Moore,[12] Thomson[15] and others have contributed to the subject.

The objective of the operation is the removal of the cancer with as little compromise of the integrity of the larynx as is consistent with complete eradication of the disease.

The story of the operation and its indications is very interesting. Originally it included section of the cricoid cartilage and was indicated for benign growths, as well as malignant ones.

Cancer cases were poorly selected, so that recurrences soon followed the operation. Many cancers, some with secondary infections, were operated upon which were not suitable for laryngofissure under any circumstance. After the operation the interior of the larynx was packed and the packing was left in for several days, a factor which favored complications; in addition, the patient was kept in the Trendelenberg or lateral recumbent position, without a pillow, for some time. These postoperative measures favored the development of pneumonia, which occurred rather frequently.

Tracheotomy was usually performed, and is still being done, by many operators as part of the laryngofissure operation. Most surgeons remove the tube on the same day or within twenty-four hours, as soon as they are certain the patient can swallow. Tracheotomy has proven to be unnecessary in our experience. It is conceivable that a patient might require a tracheotomy after

operation because of bleeding or an unusual reaction. In such a case we would proceed as follows: The cricothyroid membrane would be exposed and a tracheotomy tube temporarily placed through it. Immediately following the placement of the tracheotomy tube in this location, a deliberate classical low tracheotomy would be performed, and the upper tube withdrawn. The upper tube would be in place for only a few minutes and could do no damage. This technic is frequently followed on our service when a rapid tracheotomy is required in an emergency, especially if the operator is not certain of being able to introduce a bronchoscope or an indirect intubation tube quickly.

Until 1907 the internal perichondrium was not given special consideration. At that time J. Solis Cohen[5] suggested the removal of the soft parts, together with the perichondrium, so as to insure a more thorough removal of the diseased tissue. The suggestion of Cohen proved a marked advance in the technic of laryngofissure. It was claimed for his method that the growth could be removed in one piece, untouched by any instrument. Almost ten years later Lambert Lack suggested the removal of a part of the thyroid cartilage, together with the overlying soft tissues. This, he claimed, facilitated the exposure and removal of the cancerous area. He contended that the two halves of the larynx were more readily retracted as a result. He claimed that pain on swallowing and soreness resulted from the forcible retraction of the thyroid alae, and that they were eliminated by this procedure. We have not encountered pain following retraction of the alae. Resection of the cartilage is of value if the cancer is suspected of having penetrated beyond the inner perichondrium and into the cartilage. If we are certain that this is not the case, the cartilage resection is not done. It is the removal of the cartilage which converts the previously designated laryngofissure into a hemilaryngectomy or subhemilaryngectomy.

Indications for Operation:

Cancer, localized to one side of the larynx, not including the arytenoid areas and not extending above the ventricular band, or below level of the cricoid rim, is suitable for laryngofissure removal.

Operative Procedure:

The usual preliminary, preparatory and diagnostic procedures are carried out. The patient is given 1 grain of phenobarbital the

evening before, 1 grain six hours before, and 3 grains one hour before operation.

Anesthesia:

The interior of the larynx is anesthetized with a 10% cocaine adrenalin solution, as for direct laryngoscopy, before the patient is placed upon the table. This anesthesia eliminates the necessity for anesthetizing the larynx after the thyroid alæ have been separated, or after an opening has been made through the cricothyroid membrane for this purpose. The cocainization, which is carried out on the table, is always accompanied by struggling and coughing. A general anesthetic is used by some, but it has certain disadvantages: There is more bleeding and much time is consumed.

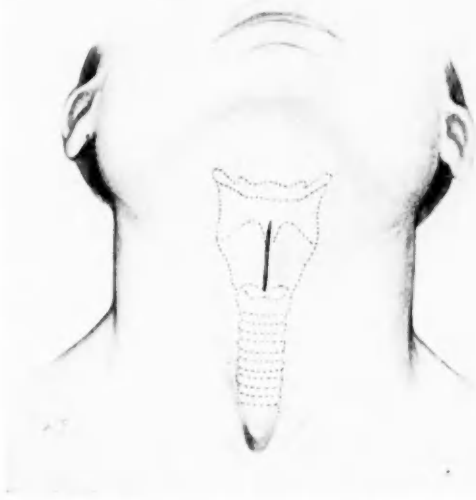

FIG. 1 — (Myerson) — Laryngofissure. Skin incision.

Incision:

Exposure:

The incision is made through the skin from the highest palpable part of the thyroid notch to the inferior border of the thyroid cartilage. This small incision is quite ample because the skin in this region is very loose and can be readily accommodated to whatever exposure may be necessary during the operation.

Dissection of the skin from the underlying structures is not done. The platysma and sternothyroid and sternohyoid muscles are not in the field of operation. The incision extends down to or through the deep cervical fascia covering the cartilage. An incision is then made in the midline through the perichondrium. An electrically driven rotary saw with a blade of such size as to cut through the cartilage but not into the soft tissues beyond is now used. The lower portion of the thyroid cartilage is thicker than the upper, so that more time and pressure may be necessary to cut through it. It is important to have an assistant drop water along the path of the saw blade to prevent searing and devitalization of the cartilage. The saw cut is rapidly accomplished and

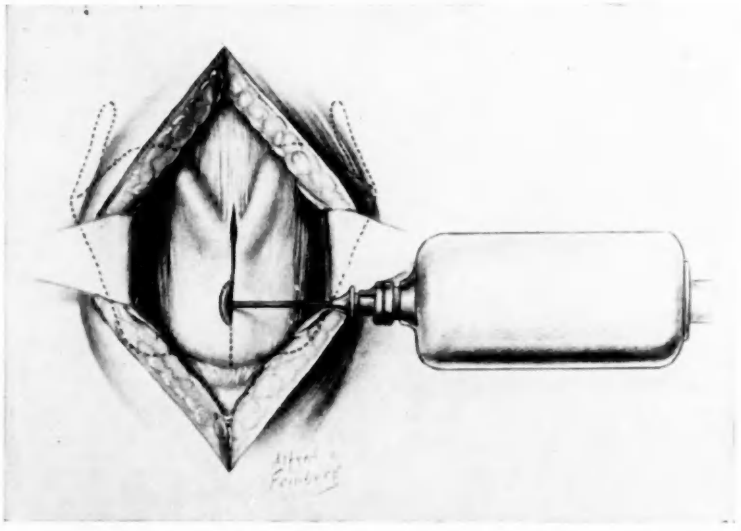

FIG. 2 — (Myerson) — Laryngofissure. Rotary saw powered by hand motor shown cutting through thyroid cartilage.

produces a wound with clean edges. After cutting through the cartilage the perichondrial elevator is used to make certain of the separation of the two halves. Then a special, three-pronged, sharp retractor is slid between the alæ, and each side is slightly retracted. The prongs are inserted between the inner perichondrium and the cartilage, and do not obscure the view nor interfere with the instrumentation. This retraction exposes the soft tissue of the anterior commissure, which is cut through in the midline. After the soft tissues are incised, a special blunt retractor is

placed directly over the lateral aspect of the thyroid notch, so that there is no interference with the perichondrial elevation. An adequate exposure of the nature and extent of the carcinoma is thus afforded. It is at this time that the operator must determine whether a laryngectomy only will suffice, or whether excision through this exposure can accomplish the desired result. He must also decide whether the cancer might have penetrated the internal perichondrium to involve the cartilage. If this is so, that portion of the ala which corresponds to the cancerous area and its immediate surrounding healthy zone will have to be excised. If the disease has not penetrated to the perichondrium or beyond it, the cartilage is not removed.

FIG. 3 — (Myerson) — Laryngofissure. Retraction and Exposure.

Removal of part of the alar cartilage was originally suggested by Lack in 1916. He claimed that it afforded easier retraction and better exposure. That better exposure is afforded by the removal of the cartilage cannot be denied; it has been our experience, however, that the additional exposure is not necessary. The amount of retraction which can be carried out when the cartilage is retained seems to be quite ample for all purposes. Lack stated that the forcible traction upon the thyroid alæ later

caused pain on swallowing. We have not noticed this to be so in our cases. The claim that the presence of the denuded area of cartilage delays healing may be correct, but this is of minor importance. On the other hand the claim that removal of the cartilage interferes with the normal relations and functions of the larynx is not borne out by our experience. There is a strong impression among laryngologists that after extensive cartilage removal, collapse of the lateral aspect of the larynx may occur. This is not so.

Excision of the Cancer Bearing Area:

When the cartilage is to be removed, a special punch is used which includes the cartilage and all the soft tissues, from the mucous membrane of the larynx to the external perichondrium. This punch cuts a rectangular section of the cartilage and laryngeal tissues which measures 24x15 millimeters. If the involved

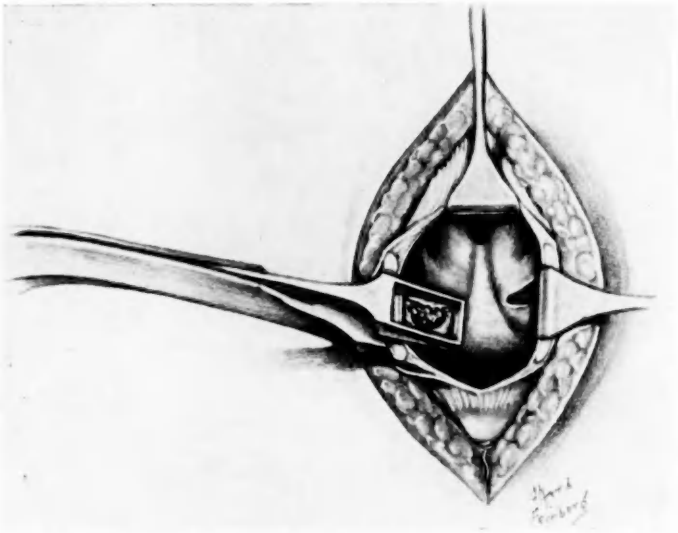

FIG. 4 — (Myerson) — Laryngofissure. Removal of Cancer Bearing Area and Cartilage in one piece by means of the special punch.

area should require a removal of tissue beyond the limitations of the punch, the cartilage is freed from its internal and external perichondria, the upper and lower margins are separated, and a special angular knife is used to make a vertical section through the posterior portion of the cartilage. Before the soft structures

are excised three hemostats are applied. A hemostat is placed horizontally at the upper and lower limit of the area to be removed before the straight scissors are used. After incision with the scissors, a right angled hemostat is applied to the posterior boundary. This is followed by excision with special angular scissors. The hemostats are left in place for two minutes before the incisions are made. The special punch may be used for removing the soft tissues.

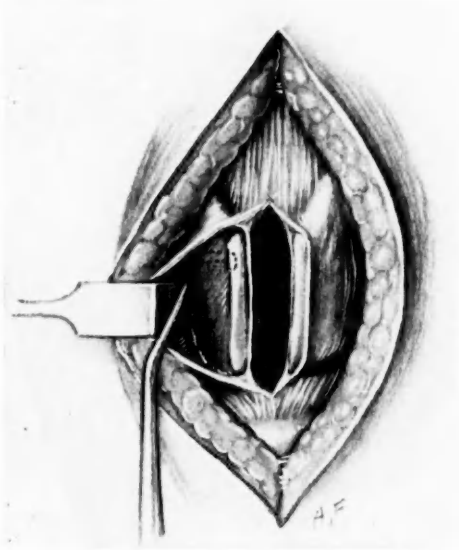

FIG. 5 — (Myerson) — Laryngofissure. Removal of part of alar cartilage by special downward cutting knife preliminary to excision of the cancer bearing soft tissues.

After the tissue is removed, there is very little bleeding. This is carefully controlled by means of small sections of dental roll held by Kelly clamps. Occasionally a vessel situated posteriorly in the severed muscle will bleed. This is readily controlled by the application of a hemostat which is left in place for two or three minutes. The branches of the superior laryngeal artery which supply the posterior part of the larynx are relatively small; therefore such procedures as ligation of this artery or the placement of suture ligatures do not seem to be necessary.

At times the upper part of the ventricular band, or an overhanging curtain of the lateral fold of the larynx above it, pro-

trudes into the air passage. It is best to excise this overhanging tissue so as to avoid interference with breathing.

Closure:

Inasmuch as only the skin, superficial fascia and deep fascia are involved in the incision, skin sutures only are required to close the wound. Two sutures are usually sufficient for this purpose. The lower angle of the wound is left open and a strip of rubber dam inserted. This prevents subcutaneous emphysema and is removed after twenty-four hours.

For cancer of the anterior commissure area the operative procedure of Jackson[10] may be used. For epiglottic cancer the technic of Tucker[17] is followed. The operation of Tucker was used in one of our cases.

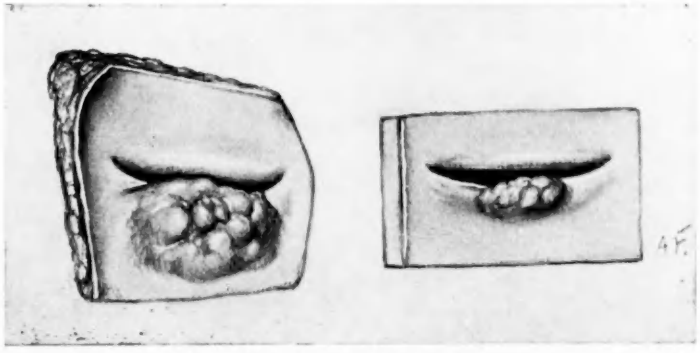

FIG. 6*a* — (Myerson) — Laryngofissure. Section of tissue excised after removing part of the cartilage with the aid of the downward cutting knife.
FIG. 6*b* — (Myerson) — Laryngofissure. Section of tissue including alar cartilage removed by the special punch.

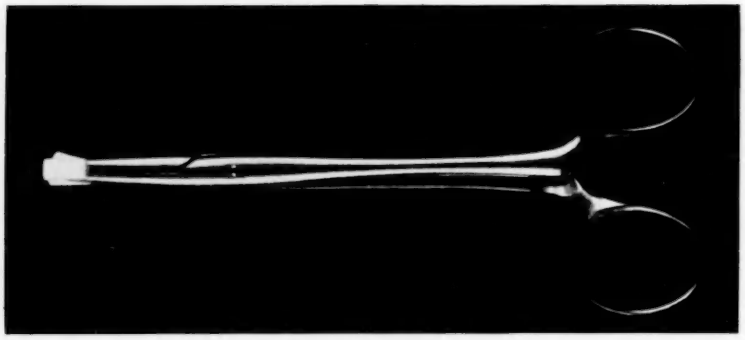

FIG. 7 — (Myerson) — Laryngofissure. Section of Dental Roll on hemostat as used for sponging operative field.

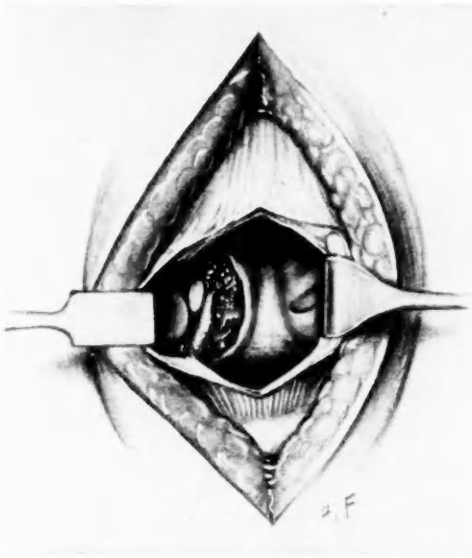

FIG. 8.—(Myerson).—Laryngofissure. Field of Operation after removal of the cancer bearing area.

Postoperative Care:

The patient is returned to bed and placed in the semi-recumbent position. Upon arrival from the operating room he is given a suppository containing one grain of codeine and ten grains of aspirin. In our experience this does not interfere with the cough reflex. If the room atmosphere is very dry, moisture should be supplied by means of a steam kettle placed at some distance from the bed. Four hours after operation, sterile water is given by mouth, in very small quantities. If the patient is able to swallow he can have larger quantities. Fluids can be given any time after the anesthetic effect has worn off. There is usually some slight pain, but the patients can swallow despite it. Patients may be permitted to leave their beds on the day following the operation or the day after. They are restrained from speaking for one week after operation. After this they are advised to speak softly and in moderation.

Complications:

Bleeding:

If complete hemostasis is established at the time of operation, postoperative bleeding does not occur. As previously mentioned,

if bleeding should occur the wound could be readily opened and the bleeding controlled. If measures other than packing would not suffice, a tracheotomy could be done. Such a procedure has not been found to be necessary in the postoperative care of more than thirty-seven cases.

Reaction of Laryngeal Tissues:

If the tissues are roughly or unnecessarily handled, reaction in the form of swelling and edema will ensue, which may compromise the airway. Under such circumstances a tracheotomy might become necessary. Tracheotomy will not be necessary if the tissues are handled carefully.

Subcutaneous Emphysema:

Subcutaneous emphysema will occur if the wound is sutured firmly. The lower angle should be left open, and a small strip of rubber dam inserted to permit the air to pass to the outside instead of into the fascial planes. After twenty-four hours the rubber dam can be removed, because at the end of this time these planes are obliterated by the healing process.

Granuloma:

Granulation tissue occurs in all cases of laryngofissure; in some more than in others. The mere fact of its presence is not an indication for its removal. It is exceedingly rare for a granuloma to become dangerously obstructive. Granulation tissue is a normal product of the healing process and eventually disappears. It is better not to disturb it unless it interferes with phonation or causes obstruction. One must differentiate between a granuloma and the reappearance of a cancer. If the mass is shrinking, one can be sure it is a granuloma; if it is increasing in size one must suspect cancer. When cancer reappears during the postoperative period, pain in the larynx or pain referred to the ear will be present, as a rule.

Chondritis and Necrosis:

This is uncommon except in cases which have been subjected to x-ray therapy. Attention to the placement of the retractors is important in the prevention of chondritis and necrosis.

Recurrences:

The extensive resection of cancer of the larynx by the laryngofissure approach is comparatively recent in this country.

Experience will enable us to determine how extensive a lesion can be safely and successfully operated by this method. We have set the following as the greatest extent of a cancer which is suitable for laryngofissure and excision:

1. Limited to one side:
2. No involvement of the arytenoid area.
3. No involvement above the ventricular band.
5. No involvement below the crest of the cricoid cartilage.

CONCLUSIONS

Laryngofissure is a relatively simple procedure. There should be no fatalities or severe local reactions following this operation. Our experience suggests the following:

Anesthetization of the larynx before operation.

A smaller incision than is usually employed will suffice.

Tracheotomy is not necessary. It is conceivable that it might be required on a rare occasion. In such a case the operator would be forewarned and would perform the tracheotomy at the time of the fissure operation.

Adequate exposure can be obtained without extensive incision of the thyrohyoid membrane and without cutting through the cricothyroid membrane. Retraction of the cartilages gives a sufficient exposure of the interior of the larynx for whatever instrumentation may be necessary. Removal of part of the thyroid ala for this purpose seems unnecessary. When necessary, this removal can be accomplished without extensive dissection and exposure of tissues. Elevation of the external perichondrium and of the soft tissues attached to it is readily accomplished by means of an elevator. A long, flat retractor is then inserted, which gives ample exposure. Where the disease is fairly limited, and the cartilage is to be removed, a special punch is offered which includes in its bite the soft tissues of the larynx, together with the cartilage. If the lesion is too extensive for the punch, an angular, downward cutting knife is used to section through the cartilage posteriorly, and the soft tissues are excised. Hemostasis is obtained by the use of small sections of dental roll or small intestinal sponges on the ends of fine hemostats. When ordinary pressure with these will not suffice, the application of a hemostat to the bleeding point will control the bleeding. Suture ligatures or plain ties are not used. Gentle and careful handling of the endolaryngeal tissues is essential to insure a minimum of reaction.

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DISCUSSION

Dr. Gabriel Tucker, Philadelphia: Ladies and gentlemen, it is a very great privilege to come before this society and discuss Dr. Myerson's paper on laryngofissure. Some two weeks ago Dr. Myerson sent me his paper and today before the meeting he told me he had revised it and improved it. When I read his paper, I didn't think it could be improved, but Dr. Myerson has improved his paper and I have no criticism whatever.

I think he has advised a number of things that are a decided improvement in the ordinary technic of the laryngofissure. Dr. Myerson, however, had left out one class of case in which the laryngofissure has been done by Dr. Jackson's technic, and I think it should be mentioned. When I read the paper, I thought then that I would limit my discussion to this matter

of anterior commissure growth, and I happen to have a series of photographs of a patient showing the different steps of this procedure. I may say in this series of laryngofissure cases, there have been some fifteen cases of anterior commissure growth and the recovery has been as high in that type of case as any other type.

The most important thing in the laryngofissure is dealing with the internal perichondrium, or the perichondrium. The first line of defense in cancer of the larynx is the internal perichondrium, the second the cartilage itself, and the third is the external perichondrium. In very early growths where the internal perichondrium is not involved, certainly it is not necessary to remove the cartilage. If the cartilage is involved, if it has gone through the internal perichondrium, you must remove the cartilage.

We must give Dr. Chevalier Jackson and Sir St. Clair Thomson the credit for popularizing the technic and the operation of laryngofissure. Twenty years ago when my association with Dr. Jackson began, there were very few laryngofissures performed, but due to his teachings and his results and those of Sir St. Clair Thomson, you know how common the procedure is today and how many larynxes have been saved.

This was a gentleman about sixty-two years of age who had an anterior commissure growth. The cords and the anterior commissure were involved. We were certain it didn't involve the thyroid membrane. The preparation was as Dr. Myerson has outlined, except that we give morphine an hour before the operation. One per cent novocaine is used and the incision is made from the hyoid bone just before the cricoid, through a portion at least of the thyroid sufficient to give an exposure to the trachea. We do not do away with the tracheotomy ordinarily but there are times when we are glad to have this preparation ahead, and this was one of the cases we found before we got through.

This is the exposure of the front of the larynx. Ligation of all bleeding vessels was carried out.

The internal perichondrium is separated at the thyroid notch, and also below, from the cricothyroid membrane. The incision is carried out from below and above separating the internal perichondrium before the larynx is opened. The anesthesia within the larynx is carried out by applying ten per cent cocaine in the larynx itself. There may be some advantages in Dr. Myerson's method of cocainization prior to opening the wound in the neck, but this has the advantage of having your cocainization at the time you need it, and we haven't had any difficulty with the patient struggling while we were putting the cocaine in through the incision in the cricothyroid.

This shows the opening of the thyroid cartilage with the curved saw, and then the wings of the cartilage are separated, being careful to separate the internal perichondrium on either side. Of course, you must not invade the internal perichondrium.

After it has been separated, a knife is used to cut through so that you can see just the extent of the growth before any incision is made. The internal perichondrium which stretches across from one wing of the thyroid to the other is dissected and that wing is discarded. Then the incision is carried out on either side. It may be necessary — and Dr. Myerson's technic is the same as we use — to use a hemostat and cut inside the hemostat, keeping the hemostat well outside the growth on either side. Ligation of bleeding points is carried out.

Where there is extensive excision in the anterior commissure, the epiglottis has a tendency to drop inward and cause obstruction to breathing. To overcome this, we have used what we call a suspension suture. This suture is placed just above the wing of the thyroid cartilage and out through the skin and it is carried from the inside on the opposite side and then the suture is pulled together, and in that way this suture holds up the base of the epiglottis.

The men whom I have talked to who have done this operation say they have been unable to manipulate their patients. As yet we haven't had one we couldn't manipulate after this procedure of removing the anterior commissure. After all, the patient is better off with his larynx than he would be with a laryngectomy tube and no larynx, so we feel the removal of the anterior commissure is justified.

In this patient, as we always do, when we got through we closed the incision and found he couldn't breathe through his larynx, so we put a tracheotomy tube in below the cricoid and you see where the incision was made. The reason he couldn't breathe was because he had spasm of the larynx. We knew this man was that type of individual, because when we do laryngectomies we have a great deal of trouble. There was no reason why he should have had spasms, so we put in a tracheotomy tube and left it for twenty-four hours and his recovery was uneventful.

Now where complications develop: in our experience it has been due to infection in the wound. You are dealing with infection in the tracheobronchial tree and when the patient coughs you have that infection in the cartilages and under the skin, and if you get it you can take out the stitches and leave it wide open and let it heal by granulation.

As I say, the results in this particular operation are as good as they are in other cases where the other type of operation is used. Certainly there is no reason why you shouldn't save the larynx. If you have a recurrence, you can still remove the larynx by laryngectomy if you desire.

DR. JOSEPH C. BECK, Chicago: As usual, when Dr. Myerson presents a paper, as he reads I make notes and then he comes back and mentions that which I thought he was going to leave out, so it doesn't leave very much to discuss.

I didn't see the paper. I just had a few moments' talk with Dr. Guttman for whom I am apologizing. He went over to check on Czechoslovakia, and to save it, I guess. I don't know what he is there for, but I am here to discuss this very interesting subject. I wish Dr. Myerson had reached a period of life in his laryngofissures where he could give us statistics as to how long after these operations these patients live without recurrence.

I also wish he had mentioned a little bit — even though it is just technic, it is the technic that is depended upon — about the type of cancer that he is operating on. He mentioned the indication as to the location, but that is not all. What type is it? It may be located where he said it was and yet be absolutely inoperable because it is a radio-resistant tumor that will play havoc with a laryngotomy or laryngofissure, or any other kind of operation. And so it is quite important to bring these points up.

With a slight movement of his hand he said Ohngren's technic of ligation is just too much for him. It isn't too much for me, and if I were operating on a case it would control hemorrhage, for that is the thing that bothers in these cases, especially where you do not have tracheotomy. Even for a skilled bronchoscopist, it may control the bleeding.

Dr. Lederer called attention to ligation and said, "What is the trouble with preliminary ligation of the arch?" and of course it is a very excellent point that I think is not to be discarded. It isn't so difficult to do that. Unfortunately, I haven't had cases that were so manifold as to require this treatment, even incision of the cartilage. I have done a few. I am still filled with wonder at the marvelous results of Sir St. Clair Thomson in the excision of the cartilage for cancer, but most of the cases that I see come too late for that. Of course, as Dr. Tucker said, it is better to have a part of the larynx than to have the larynx out, but I think the life of the patient is more important, that is what they come for when they have cancer, and that means that is the job, to get rid of the cancer. I think that comes first, before the voice or anything else.

The anesthetic Dr. Myerson mentioned is just a local anesthetic. I think your patient is so much more tranquil and quiet with a rectal type of anesthetic. You don't have to have an anesthesia that is going by the airway, and rectal anesthetic has been a great aid in our work on the larynx, both laryngofissure and laryngotomy.

I think this operation is going to progress. As Dr. Myerson said, the early cases were failures because the men didn't know how to do an operation so far as saving the tissues was concerned. They were breaking the cartilage.

The saw that was mentioned which Dr. Guttman first presented to me —I don't know whether it was original with him or not — is wonderful in those cases where the cartilage is friable and breaks. Any other method is difficult and it is an excellent thing, as Dr. Myerson mentioned, in cutting through that cartilage.

And then I wonder why Dr. Myerson forgets about surgical diathermy. That is another point Lederer said was the matter with that. (I didn't know what I was going to say until Lederer had told me what to say.) But it is so. Surgical diathermy is many times very delicately used, not destroying the cartilage, and not using it to the extent that you would in perichondritis secondary. That is not necessary. With the soft tissue, you control the bleeding and you prevent further implantation, and surgical diathermy in a distinct technical point in laryngofissures.

I congratulate Dr. Myerson for bringing up this subject of laryngofissure again.

Dr. M. C. Myerson, in closing: Gentlemen, I am sorry that I omitted to speak of anterior commissure growth, because mention of these growths is made in the paper. I say in the paper, and I say now, that one must expect to have put tracheotomy tubes in a good many of the patients upon whom he operates for anterior commissure growths, no matter what he does at the time of operation. I think that Dr. Tucker's suggestion is a very good one, and although I have not done it up to the present time, I intend to try it hereafter.

I also mentioned in the paper, and did not mention as I spoke, epiglottic cancers which Dr. Tucker has been able to remove by extending his laryngofissure technic.

Dr. Tucker favors cocainization of the larynx at the time of operation because he would like to have the cocainization when he wants it. Once anesthesia is established, the anesthesia certainly lasts three quarters of an hour or more and there is no reason why this operation, no matter what the operation entails, can't be done in twenty or thirty minutes at the outside. So the average time for this operation is fifteen to eighteen minutes and it could be done in less time if one were looking to establish time records, but that is not what we are looking for in surgery.

I noticed in the picture that Dr. Tucker's patient had a nasal feeding tube. We have never used a nasal feeding tube and I wonder if Dr. Tucker can explain that for us.

DR. TUCKER: We have used for a long time in any operative work on the larynx a catheter through the nose, and used sutures. Instead of packing the pharynx, we use intermittent suction. The anesthetist uses suction to keep the pharynx clear. The tube was a tube used for keeping the pharynx clean and not a feeding tube.

DR. MYERSON: Dr. Beck brought up some very vital points, and he speaks of statistics. I have no statistics except that the first laryngofissure I performed was done in 1922 and that patient is still alive. In a period of twelve months which ended, let's say, five months ago, I had done eleven laryngofissures and three of those recurred. I believe one of those was an anterior commissure growth and the other two were very extensive subglottic lesions. I make the statement in this paper that as our experience grows, we ought to be able to tell just how extensive a cancer we can tackle by this method.

The question of the type of cancerous tissue from the standpoint of the therapy indicated is not a separate question by any means. We must appreciate that when a section of tissue is sent to the laboratory, just a very small slice of that tissue is examined under the microscope, and when that slice of tissue shows metaplastic cells, let's say a Grade 4, if you will, we don't know but perhaps two millimeters beyond that tissue we might have found a Grade I type of carcinoma, and therefore it is not uncommon or unusual for us to be surprised in our clinical experiences when we encounter a case we feel is very radio-resistant, and because the patient is a morphine addict, we submit him to x-ray therapy despite the fact that he has a radio-resistant carcinoma, and to our astonishment and pleasant surprise the patient is cured by the x-ray therapy. So we cannot place too much emphasis or reliance upon the type of cancer.

I mentioned the point that any type of general anesthesia does influence the amount of bleeding, and I believe if we are going to use general anesthesia, we must routinely perform a tracheotomy. If I were using general anesthesia, I think my technic would include tracheotomy routinely.

I am glad Dr. Beck mentioned surgical diathermy because we do use surgical diathermy where we believe the cancer is more extensive than we expected, and we burn it out before we have done our laryngofissure.

* * *

DEVELOPMENT OF THE COCHLEA

RALPH A. FENTON, M.D., for
OLOF LARSELL, Ph.D.
PORTLAND, ORE.

DOCTOR LARSELL desires me to express to the Academy his gratitude for its support of his research during the year.

This research, when completed, will fill out the details of development of the mammalian cochlea as previously studied by Dr. Larsell and Dr. Macready on the pouch-contained embryo of the opossum. It was found by watching body and limb movements that the high tones were perceived two weeks earlier than low tones; and this was checked by Drs. Wever and Bray through electric magnification of action currents, after which the stage of development of the cochlear nerve terminals was checked by serial microscopic section.

The research now in progress relates to damage of the cochlea at various levels and various ages by electrosurgical methods, following which the movement and electrical responses were tested. Microscopical examination of these injured cochleas correlating these factors is now in progress, and it is hoped may contribute materially to our understanding of certain problems relative to congenital or to early acquired deafness.

Final report will be made at the 1939 session of the Academy.

* * *

WALTZING GUINEA PIG

M. H. LURIE, M.D.
BOSTON

IN THE past four years at the Harvard University we have been studying a special breed of guinea pigs called circling or waltzing guinea pigs. These animals are similar to the waltzing or Japanese dancing mice. They circle in either direction, right or left, and do so especially when excited or hungry. Some animals have a tendency to circle chiefly in one direction. The majority seem to circle in the direction in which they happen to start and will not reverse until that period of excitement has stopped. They show no nystagmus and the righting reflexes are normal. The head has a fine tremor most marked when it is extended. The animal is deaf and does not respond to any external sound stimulation. All these characters are inherited and follow the Mendelian laws for recessive characteristics. The animals are difficult to raise and to breed. To maintain this strain, they must be bred with normal guinea pigs and the offspring then re-bred to obtain the strain back again. All the hybrids are normal.

In all animals studied up to the present time no pathology can be demonstrated either in the medulla or the vestibular portion of the ear.

The utricle, saccule, semicircular canals, scarpas ganglion, vestibular nerve and vestibular nucleus, are normal by histological study.

Slides

The circling is not caused by the vestibular apparatus but by some change in the central nervous system not yet found.

Deafness in these animals is the result of degeneration of the organ of Corti and the cochlear nerve. The degeneration takes

place in the same manner in all the animals studied. It begins at the first turn and extends toward the helicotrema .In none of the animals except a few of the older ones has there been a complete absence of the organ of Corti. There is always present some normal or almost normal organ of Corti at the helicotrema. A response by the Wever-Bray method can be obtained for low tones with high intensities from this portion of the organ of Corti. The degeneration begins in the organ of Corti first and the cochlear nerve secondarily. At the present time we believe this degeneration starts after the birth of the animals, and, therefore, is considered as an inherited type of deafness rather than congenital.

Slides

These findings raise the question — can perception or nerve deafness be inherited? The study of these animals shows that nerve deafness can be inherited. This may explain the nerve deafness found in children and young adults who have had no disease to explain the deafness.

This hereditary factor may also explain the reason for a unilateral deafness. We do know that animals with inherited deafness can have a normal ear on one side and a completely deaf ear on the other. This has been found true in cats and dogs.

The study of these interesting animals has helped to solve some of the problems in the physiology of hearing, but it has also added more complications to the understanding, treatment and prevention of deafness.

Panel Discussion

SINUSITIS

CHAIRMAN ERNEST M. SEYDELL, Wichita, Kans.: I am certain that it is a very common experience with all otolaryngologists in the examination of a new patient when he makes the usual inquiry, "What is your complaint?" to receive the answer, "I have sinus trouble."

We have this afternoon a hypothetical patient who has made this answer. The members of the panel will now conduct the examination of this patient largely as they would in their own offices, and will follow the necessary procedures of thought and action so that ultimately a correct diagnosis will be made and the proper method of treatment instituted.

Dr. Ridpath will now discuss the question of "Taking the Patient's History and Examining the Patient."

Panel Discussion

SINUSITIS

* * *

Taking the History and Examining Patient

ROBERT F. RIDPATH, M.D.
PHILADELPHIA

I FEEL we are all agreed that a carefully compiled history of a patient taken before and during the examination is a necessary essential to completeness. I feel that history taking should be a personal matter between the examiner and the patient. In my office (and I think we should speak personally in this discussion) my secretary brings in the history card with the date, the patient's name, age, occupation, residence, and who, if anyone, referred him. The rest of the history is then compiled by myself. It brings one into a confidential and intimate contact and results in a decidedly more comprehensive and illuminating history than can otherwise be obtained.

One of the first questions I ask is "Where is your pain?" Not "Do you have pain?" but "Where is your pain?" If the answer is "In my head," then the suggestion "Put your finger on the spot of greatest pain." This gives some idea of location and leads to the next question, 'Is the pain constant or only present at intervals?" "How many *days* have you had it?"

These questions are then followed up by the mode of origin.

"Did it come on suddenly?"

"Did it result from a cold?"

"Did it come from other infectious diseases?"

"Did you have an accident?"

And then the character of the pain:

"Is it sharp and stab-like?"

"Is it dull and sickening, etc.?"

"Is it throbbing (pounding)?"

We now concentrate to local areas, such as:

1. Maxillary sinus — Supra orbital notch, teeth, cheeks, etc.
2. Ethmoid, band about the head, between eyes, temporal region and fronto-parietal.
3. Frontal — Eyebrow to hairline.
4. Sphenoid — Eyeballs, occiput.
5. Mastoid — Acute otitis media, mastoiditis, sphenoid.

Then "Is the pain worse at night?"

"Is the pain worse in the daytime?"

"Does it occur at anytime?"

People like to talk of their pains and from my experience, it is the easiest way to gain their confidence and interest to begin our history and our examination by permitting them to talk freely until this symptom is fully described.

CHAIRMAN SEYDELL: May I interrupt to ask a question? Is it not possible that myalgia may produce pain similar to any of these described?

DR. RIDPATH: Yes, myalgia simulates pain of a sinus origin in many instances.

DR. HARRY P. SCHENCK, Philadelphia: You mention pain being in the eyeball or in the occiput in sphenoid disease. I happen to know of two cases on which I had to operate in which over a period of six months very frequently I was able to reach the sphenoid with a probe, and in one case he always placed the pain in the occiput. In the other he always placed it in the vertex.

DR. RIDPATH: Pain from the sphenoid sinus happens to be many places throughout the head. We have the direct pain from perhaps a toxemia and we have so-called reflex pains from the sphenoid. I mentioned the pain in the eye and the occiput. The pain in the eye is characteristic in sphenoidal sinus infection because it gives a pain and a pressure along with the pain back of

the eye, simulating the pushing of the eye forward. This is not always present in a sphenoid infection, but it occurs so frequently that it is a cardinal symptom.

Essentially, the one pain which is spoken of particularly in the books in regard to sphenoidal infection does occur in the occiput and radiates in fan-shape fashion up to the vertex.

I think pain over the mastoid area is probably just as frequent as the pain in the eye or the pain over the occiput.

The second important subject is discharge. I like to ask, "How many times do you use your handkerchief in a day?" If they use more than one handkerchief there is a potential sinus infection.

DR. SCHENCK: Isn't it true that allergic individuals may use a great many handkerchiefs during the course of twenty-four hours?

DR. RIDPATH: Very true and in vasomotor rhinitis we have the same answer. I used the word "potential." We have a potential sinus infection until the examination develops the allergic condition or a vasomotor rhinitis, so I am going to adhere to my original thought, namely, anyone who uses over one handkerchief a day has a potential sinus infection.

DR. ANDREW A. EGGSTON, New York: Is there any attempt at collection of the discharge for examination by miscroscope to determine whether it is allergic?

DR. RIDPATH: Later in my paper I will mention that if the picture presents characteristics in which we feel that we should have a microscopic examination made to determine the offending factors, essentially we make a culture.

DR. EGGSTON: I had in mind a gross and serous type of discharge.

DR. RIDPATH: I will come to that in my paper. I repeat, if they use more than one handkerchief there is a potential sinus infection. Should the answer be "Why, I never use a handkerchief" the next question is: "Do you cough or expectorate any material from your mouth or throat?" If the answer is in the affirmative the same assumption may be had. Having had an

affirmative answer we follow with, "Is it watery?" "Creamy?" "Scabby?" "Jelly-like?" etc.

The third division of the examination should concern impaired breathing and with questions which will give us the following information. "Can you breathe equally well through both sides of your nose?" "Is this of recent incidence or is it long standing?" "Is there any difference in your breathing night or day?" "Is the condition made worse by (a) posture (b) seasons (c) weather conditions (d) heat or cold (e) inhalation of (1) dust (2) tobacco smoke (3) pollens (4) chemicals (5) occupation and position or environment in which you work?"

The fourth subject would be vertigo:

"Its frequency, duration?"

"Is it momentary?"

"Is it lasting?"

"What in your mind contributes to the dizziness?" "Does it come on when you stoop forward, when you read, when you lie down or it is liable to occur at any time?"

Certain characteristics of the patient should be noted during the history taking and examination. It is rather accepted "that to be a specialist one must first be a good physician."

Dr. Richard Kern, Philadelphia: You said to be a specialist a man must first be a good physician. As a former specialist in the diseases of the skin and its contents, and more recently as an internist who still has to cover a lot of territory, I would like to amend that statement a little by way of emphasis: that to be a good specialist one must first be a good physician.

Dr. Ridpath: Doctor, I accept your amendment.

Many cases give the observing clinician untold symptoms of systemic disturbances if we only look for and know them when we see them. It, therefore, behooves us to observe if our patient is anemic, plethoric, alcoholic, phthisical, gives symptoms of arthritis, syphilis, has a yellow soft palate (always indicative of gall bladder disturbance) notice the eyes, the sclera, the conjunctiva, clarity or opaqueness, the shape of the nose, nostrils and their medial or angular position. Remember the symptoms of

hypertension and nephritis since many other conditions often have symptoms simulating those of sinus infection.

We now consider the mouth and throat and the condition of the teeth, tongue, hard and soft palate, the fauces, tonsils or fossa, the pharynx and lingual tonsil. The glands on the floor of the mouth should be palpated. Post-rhinoscopically we note the posterior end of the septum, inferior turbinate, middle turbinate, superior turbinate, the vault and if possible the Eustachean orifices. It is during this examination that the pharyngoscope should be used and the more one becomes accustomed to its use the more he will see.

By the use (and this should always be included in our examination) of the laryngoscope we view the larynx and note its position and size, whether it is red or anemic; the arytenoid cartilages, the ventricular bands and cords; the shape, condition and anatomical relationship of the epiglottis is a special point to remember. By this means the trachea in its upper part may in the majority of cases be visualized.

I feel we should also have a working knowledge of the ophthalmoscope. Transillumination is to be considered from the knowledge one may deduct from its use and also the psychological effect on the patient. Should a questionable diagnosis be at issue we then may use: X-ray, lavage with or without an opaque fluid. I have little sympathy for the rhinologist who is unable to make a diagnosis of his case without the use of the roentgenologist, but his aid should always be considered before an operation, but to permit or to look to the X-ray man to make our diagnosis for us is unjustified and shows lack of training and knowledge on the part of the rhinologist.

Cultures from the nose and throat should be taken and a careful blood examination made. Blood pressure, the urine, a study of the spinal fluid — all enter the picture, if our history and other subjective or objective symptoms warrant.

CHAIRMAN SEYDELL: We have completed taking the history of this hypothetical patient and we will now go on to the next subject, "Roentgen Ray As a Diagnostic Aid," by Dr. Alexander S. Macmillan of Boston.

Panel Discussion

SINUSITIS

* * *

Roentgen Ray as a Diagnostic Aid

ALEXANDER S. MACMILLAN, M.D.
BY INVITATION
BOSTON

WHEN this patient is referred for Roentgen Examination, I would make four exposures — three with the patient lying down and one sitting up. From these four I would expect to get as much information as could be expected from the Roentgen Examination.

The first slide that I will show is one that is taken with the patient lying on the side and the tube in such a position that I can get a good lateral view of all of the sinuses.

In this lateral position, one must include enough of the skull to rule out an obvious brain tumor which may be the cause of pain along the distribution of the fifth nerve. We may find convolutional atrophy, enlargement of the artery grooves, separation of the sutures or evidences of pressure on the sella turcica which indicate increased intracranial pressure. Furthermore, we learn the depth of the frontal sinus, the thickness of the front and back wall and any anatomical variations such as supraorbital recesses. From this position we get a good view of the sphenoid, its depth and density as compared with the other sinuses. The condition of the ethmoid cells may also be seen — whether they are decalcified or increased in density. The nasopharynx is shown up to good advantage and the presence of pathology causing referred pain to the nasal sinuses.

Slide — In this plate, we can make out what we consider to be the normal amount of tissue and through the broad air shadow may be seen the opening of the eustachian tube into the pharynx.

We can see from this slide that the patient has very large posterior tips of the inferior turbinates. Such a film as this, enables one to note impacted teeth and the premaxillary region where cysts so often occur.

In this next film of a young patient we see a definite mass of adenoid tissue, the broad band of air stops abruptly at this point, due to adenoid tissue blocking the choanae; the patient's jaws are seen to be separated, and a column of air can be seen between the tongue and the roof of the mouth.

This next slide, still in the lateral position, shows a choanal polyp which is not attached to the posterior pharyngeal wall but hangs down over the soft palate; to view this growth one would have to retract the soft palate or use the nasopharyngoscope.

The importance of the lateral position cannot be overestimated because without it several very obvious abnormalities could not be diagnosed. Take for instance, this last slide of the lateral view; we see a very small but definite mass which has grown from the nasopharynx upward into the sphenoid and has destroyed the posterior clinoidal process.

The forehead and nose position, called the Caldwell position, is a very important one and is often the only one taken, I am sorry to say. It is one in which we expect to get a good deal of information in cases of frontal and ethmoidal disease. Consider this frontal sinus: there is very little increased density as compared with the one on the other side, although this is all we expect to find in an acute frontal sinusitis; the outline of the sinus itself is very clean-cut and there is merely a suggestion of lack of contrast between the two, which is more noticeable in the plane of the supraorbital recess.

Slide — This patient came in with oedema over the forehead, causing the closure of both eyes. The density of the frontals is equal. The very black area at this point is produced by extremely thin anterior walls and this more dense area is produced by shallowness of the frontal. The only clue as to which frontal is involved, is this line of thickened membrane which is the membranous septum between the two frontals; it is pushed over by pressure from pus within the right side.

Slide — Continuing with the same position we see slight density in the frontal sinus on the right side. What can I say about that? What would your Roentgenologist say about it? He might

use the term "cloudy," "hazy" or "veiled." To you those are meaningless terms but that is about all one could say from this one view. The information you are after is the presence or absence of pus and the condition of the membrane. This brings us to the sitting-up position.

Slide — Here we see something more definite, a horizontal line half way up in the frontal which is the fluid level. The Roentgenologist can now say the frontal is half full of pus and the membrane so many millimeters thick.

Slide — This slide is a photograph of the patient in the sitting-up position. You observe the patient's head is firmly held by a holder, the chin is resting on a support and the cone comes in contact with the patient's head from behind. The ray is horizontal. On the one film we are enabled to see the fluid levels in the frontals and antra.

Slide — Note the fluid level in this frontal: There is very little pus present but the clinical symptoms are very definite.

Slide — Consider these frontals: We see one continuous line extending from the right to the left. You will note the antrum on the left side also has a fluid level while the antrum on the right is clear. Obviously we have a very large left frontal extending to the right of the midline and an extremely small right frontal. You will note in these last few slides of acute frontal sinusitis that the density of pus and thickened membrane is not very great. The story of chronic sinusitis is different. Here we have something more than the density of pus and thickened membrane and here, parenthetically, let me say that pus, mucous membrane, and normal secretion of the mucous membrane, all have the same density as far as the X-Ray is concerned. However in chronic sinusitis, we have a reaction in the bone. We have an osteitis in the bone of the frontal wall and the posterior wall, and also in the bone above the outline of the frontal sinus. That is not all! When the ethmoids and antrum are involved we have a reflection of this osteitis in the greater wing of the sphenoid, and the superior maxilla and the malar bone.

Now, this orbit in this slide is dense — not because of a cellulitis or an orbitus abscess, but because of the density in the bone, and this frontal is dense not because of pus — the other slides showed frontals full of pus — this is dense because of the reaction in the bone itself.

Slide — Here again was a unilateral chronic sinusitis.

Question: "Doctor, how old is this patient?"

Dr. Macmillan: "I don't recall but all except the 3rd molars are erupted and, from their size, I would judge the patient to be approximately fifteen years old."

Question: "Do you believe this individual has only one frontal?"

Dr. Macmillan: "Dr. Mosher teaches that the frontal sinus is never absent so we must regard this small area as an underdeveloped one."

Dr. Ridpath (pointing): "There seems to be two lines in this area. Mightn't it be possible you have a very high and very shallow frontal in this case?"

Dr. Macmillan: "We have three other films to check this point. Three other views will help decide whether the frontal extends to here or here which demonstrates the importance of the irriduceable minimum of four positions in the Roentgen diagnosis of sinus pathology. One position by itself could not be conclusive."

Slide — Here we have a case in which there is a chronic sinusitis on one side, with a markedly increased density in the bone, and a mucocele on the other side. We have as much secretion on the right as on the left, but on the side with the mucocele there is no infection and the bone is actually thinned by pressure, while on the other side the chronic infection has produced a marked sclerosis of the bony wall.

Slide — It is possible, of course, to pick out infections confined to small groups of ethmoid cells. Here we see the anterior cells, very dense and the large posterior cells, perfectly clear. However, as a rule, both the anterior and posterior ethmoid cells are infected.

Slide — In considering the ethmoid infections, let us go back to this lateral view. We see here a definite sclerosis of the ethmoid cell partitions such as is found in chronic, purulent ethmoiditis. In chronic, polypoid ethmoiditis the cell partitions are decalcified.

Slide — Here is a film brought in by a patient. Just one film with a diagnosis of sinusitis. There is no sinusitis present. The incorrect diagnosis is the result of an incorrectly positioned film.

As you see, the patient does not appear to be looking straight ahead but sideways, and we see more of one ethmoidal labyrinth than of the other. The density of the antrum is due to the projection of the external occipital protuberance through it.

There are technical errors such as this which the trained Roentgenologist is bound to recognize; such things as over and under exposure and artifacts due to errors and accidents in the dark room.

Slide — Let us now consider patients showing chronic rhinitis or sinusitis of allergic origin. At this time of year, fully half of the patients we see in the clinics are those suffering from the effect of pollens. The general appearance is one of lack of contrast — there is no cleancut detail or blackness to the entire sinus area. The ethmoid cell partitions are entirely decalcified. You can see the malar bone and zygomatic arch too clearly through the antrum. The ethmoids and antra are filled with polypoid membrane. There is an increase in the blood supply to the part and pressure from the polypoid membrane, so doubtless there has been a decalcification of bone. The condition is limited to the sinuses but it gives the entire film a milky, washed, non-contrasty appearance. Consider the front view of the same patient.

Slide — This condition is bilateral as it always is. The ethmoid labyrinth is of homogeneous whiteness, the nose is completely blocked, the antra have no contrast; it is possible to see the detail of the base of the skull projected through; there is no reaction in the bone such as we see in subacute or chronic purulent sinusitis. The frontal sinuses contain no air and the outline of thickened membrane cannot be made out, indicating the sinus is completely filled to the exclusion of air.

Slide — This is of a patient in the early stages when the condition is limited to the nasal chambers and we have an allergic rhinitis. You see the nose is completely blocked but there is good contrast in the sinuses.

Slide — Here is a patient with a more advanced condition. We begin to see the decalcification of the ethmoid labyrinth without changes in the rest of the cavities.

Slide — And here is perhaps the most characteristic picture of all: One frontal sinus is half full of secretion, yet the membrane is not particularly thickened. This lack of drainage is due to blockage

from below. Both ethmoid labyrinths are filled with polypoid membrane and secretion; both antra are half full of secretion and the thickness of the membrane is contrasted with the air content.

The last position I wish to discuss is the Waters Position, modified in that the mouth has been opened and through it we see the sphenoid projected.

Slide — In this film we are able to compare the density of the sphenoid with the density of the antrum and it is this comparison between one sinus and another, the quality of the plate, and the density of the patient's bone, that we rely upon in interpreting sinus films and not in trying to picture, or carry in our minds, one normal to fit all cases. Here is the nasal septum and it corresponds in this particular patient very nicely with the septum between the right and left sphenoid. The sphenoid goes back this far on one side and only to here on the other side. Compare this projection of the sphenoid with the lateral view, and you get a fairly good idea of its size and condition. In this view the sinuses are perfectly clear.

Slide — Here we have a film where other factors are taken into consideration. You notice the turbinate on this side is swollen and the sphenoid is not as clear as the one on the other side. This is due to the overshadowing by the enlarged turbinate. There is one part of the sphenoid which is not covered by the posterior tip of the turbinate and we see the sphenoid is perfectly clear. Recognizing this shadow as the turbinate, we discount the apparent density of the sphenoid. The rest of the sinuses are perfectly clear.

The next two or three slides will illustrate the error which is frequently made when transillumination alone is depended upon. Here we have a normal antrum but one which is very small, with thick heavy wall; on the other side is a large pneumatic mastoid.

Slide — On this side we have a small antrum with thick heavy bone and on the other side a much larger antrum with markedly thickened membrane. Both transilluminated evenly.

Slide — There is another condition where transillumination may be misleading — namely in the ordinary retention cyst of the antrum. I will show you one of these later. If you want to inject lipiodol into the antrum to show the thickened membrane, it is perfectly all right to do so. If the antrum is full of pus, what difference does it make how thick the membrane is? If the fluid level

position is taken you can note the thickness of the membrane in contrast to the air bubble. In other words you can contrast the thickness of the membrane with the air without injecting lipiodol.

Slide — About 20 per cent of the patients coming in complaining of sinus trouble have mucous retention cysts in the antrum — they are very apt to be bilateral. This is a unilateral one. You can see through it perfectly well. It almost completely fills the antrum. In transillumination that antrum would be brighter than the one on the other side. Contrast this picture with relatively acute sinusitis — even there we have a reaction in the bone which is entirely missing in the one with the cyst.

Slide — Here is one with lining membrane about 1 mm. thick that can be seen on all of the walls. This film was taken in the prone position. One would be tempted to say there was no pus in this antrum; however, the erect position shows a very definite fluid level and there was at least a dram; in this position the floor of the antrum slants back quite abruptly. In a report of "no fluid," determined solely from the other plate, and pus was obtained on washing the antrum, you could say that pus was present but did not show on the film. It is very essential to know the time interval between the X-ray studies and the current diagnosis, because the condition within the sinus may have been under treatment and might change completely in a very short time. It is like expecting to get the up to date news from last week's newspaper.

There are those patients with an antrum very dark on transillumination who give a history of recent infection. They may have pain in the face or other symptoms pointing to a suspicious antrum. The radiograph, however, shows a normal mucosa but a definite residual sclerosis in the bone which might last for weeks.

Question — Dr. Ridpath: I would like to know what your opinion is of treating sinus infection by your specialty?

Answer — Dr. Macmillan: There is an old erroneous saying "Once a sinus, always a sinus" and there are people who are surprised when a sinus clears under X-ray treatment. They do not seem to realize that such a sinus could be cleared by any other form of treatment or by the element of time. I am not particularly impressed with the reports that have come out on X-ray treatment of sinusitis.

Question — Dr. Ridpath: I noticed in one of your slides you used lipiodol. Can you interpret better using lipiodol in the almost solid form, as it is received, or have you used lipiodol diluted with poppy seed oil to 50-50 per cent? If you have, what is your thought in regard to interpretation, in regard to the mucous membrane and other characteristics of the sinuses as you see it?

Answer — Dr. Macmillan: Of course, in practically every case, you can notice the thickness of the membrane in contrast to the air in the sinus. If you inject diluted lipiodol you are going to see polyps or cysts extending into its dense shadow. We take all four views routinely without lipiodol. We have used it enough to feel that it is not necessary. There are many more positions that could be taken, and stereopic films may be made, but as a routine measure, if the lateral view, the forehead and nose position, the Waters position with the mouth open and the one erect position are taken and interpreted properly, there can be little chance of error.

Chairman Seydell: In order thoroughly to understand sinusitis, or other conditions which resemble it, it is necessary that we have a comprehensive knowledge of the underlying pathology. Dr. Andrew A. Eggston, the Pathologist for many years of Manhattan Eye and Ear Infirmary in New York, will now take up Pathology.

Panel Discussion

SINUSITIS

* * *

Pathology

ANDREW A. EGGSTON
BY INVITATION
NEW YORK

MR. CHAIRMAN, after reading the programs of this meeting I almost thought I was at a pathological rather than an otolaryngological meeting, which to me is a sign of great progress in this specialty. After attending some of the classes, I am afraid there is very little which I can add, but will review some of the observations which we have made at the Manhattan Eye, Ear, and Throat Hospital.

Probably if pathological examinations were made on some of the cases Dr. Ridpath talked about, and with which Dr. Macmillan disagreed, that perhaps a correlation of the clinical signs with the pathology at the time of operation would clarify many of the disputed points. I am perfectly frank to admit that sometimes the pathological findings are not of sufficient degree to justify the clinical results following treatment. I believe the tissues are so delicate that symptoms occur from very little pathological changes in the sinus. Thus clinicans and pathologists should correlate their findings, and therefore the surgeon when operating upon the sinuses should always send some of the material to the laboratory for routine pathological examinations. Thus valuable information would be obtainable and also would help to train pathologists in otolaryngological pathology. General pathologists have little training in special pathology of the ear, nose and throat, and they should be supplied with material for the pathological training of young men in this specialty.

One other thing I would like to suggest is that when pathological material is removed, the source of the specimen should be

indicated, as from the antrum, if you are in the ethmoid, label it from the ethmoid, and so on. Thus the pathologist may know the source and then intelligently write up a report. Information thus obtained may be of value to you and the patient. If soft tissue is sent to the laboratory, a report can be rendered in a short time. On the other hand, if bone is mixed with the soft tissue, it has to be decalcified, and the results are delayed.

Let me repeat, if you orient the tissue for the laboratory, you will get more cooperation, and satisfactory reports from the pathologist.

As the result of studying tissues that have been submitted to us I have a story to present about pathology of chronic sinusitis, that to me is interesting. Pathology is a set and sometimes dry subject, and many consider it very uninteresting and boring. When one gets into the real spirit of it and tries to explain what happens, or the process involved, pathology offers a very interesting story, second to no other one in medicine. I believe pathology should be presented on the basis of what happened, what is the meaning of this change from normal structure. In other words, try to visualize what happened to produce those changes. Only in this way is pathology a live subject.

I know most of you are not pathologists and realize it is very difficult for you to examine a slide, because you have gotten out of the practice of looking at them. However, I wish to show some slides to illustrate some of the pathology of chronic sinusitis.

This specimen shows normal turbinate tissue. You can see the numerous glands. There is very little cellular reaction. There is some vascular thickening which is relatively normal in the turbinate bones. There is no particular interest in turbinate bones, unless there is a pathological condition, a tumor, and then of course it should be examined pathologically.

The next slide is membranes taken from an antrum, and I would say it is relatively normal. The cilia are intact, there is no metaplasia, or change in the epithelium. The mucus glands are functioning rather actively. The serous glands are inactive. There is a little round cell infiltration in areas, but this is considered a normal membrane of the antrum. The antrum has a rather thick celled mucous membrane.

I don't wish to spend too much time upon the histology, but in order to interpret pathology one must know histology. Compare the relative number of glands in the turbinate bones and the

number in the antrum. The number of glands in the tissue helps to identify the tissue. There are numerous glands in the antrum, less in the ethmoids, and a few in the frontal, and a few or practically none in the sphenoid mucosa.

The next section is ethmoid and shows a lot of mucoid secretion but very little cellular reaction, which is about the normal amount of cellular reaction in ethmoid membrane.

This is a section of a relatively normal sphenoid membrane, however there is quite a lot of mucus being secreted. The mucosa shows some goblet cells; there is also a little more cellular reaction than normal.

The normal sphenoid membrane is a very delicate structure. Even at autopsy one has considerable difficulty in removing it intact. It is probably the most delicate membrane in the whole anatomy.

In the past, I am inclined to think that too much stress has been put on the epithelial pathology and not enough attention paid to the structure of the lamina propria. The stroma contains the vessels and nerves and therefore the pathology of these structures are important. The epithelium produces a great deal of objective symptoms, but the reactions, and symptoms of pain, result from injury to the lamina propria.

This slide shows the olfactory nerves in cross section. This is a reminder to Rhinologists not to fool around in the nose carelessly for fear of opening one of the arachnoid spaces around the nerves, and thus open a pathway of infection into the brain.

Acute pathology will not be considered in detail in this discussion. The next slide, however, is one of acute inflammation of a polyp and is recognized by the number of polymorphonuclears. Tissue is no different from the circulation in that the same sort of cellular reaction occurs here as in the blood, and the quantity of reaction varies, and upon that basis one can determine pathologically whether it is an acute process, sub-acute or chronic process.

Most of the cellular reaction in this slide is of a lymphocytic nature, and most of them are perivascular. With repeated attacks of inflammation this perivascular reaction produces certain permanent damages that are responsible for the chronic condition which follows.

This slide shows the perivascular reaction of fibroblasts, mononuclear cell, and lymphocytes, showing that this is not an acute process.

As the result of these perivascular changes around the veins and lymphatics, obstruction of those vessels occurs. As the result of that obstruction no drainage of the fluid from the part can take place, and a swelling and turgescence of the lamina propria occurs.

This mechanism explains the production of polyps. I have thus likened polyps to hemorrhoids. Thrombus in the veins follows phlebitis and these vessels become obstructed. Tissue fluid is not absorbed, and edema with marked distention of the tissues occurs. If the circulation in this condition is reestablished the edema will subside. In some instances polyps will disappear by proper nonsurgical treatment.

This slide reveals lymph vessels that are dilated. The obstruction may be proximal to this, that is toward the heart from this area, and you simply have dilatation distally and do not see the thrombosis.

This slide shows a recent thrombus of a vein in this region. There is a thrombosed vessel with the hyaline thrombus in the vessel not unlike you get in the largest vessels of the hemorrhoid. As a result, you have edema and atrophy of epithelium. You will notice in this region you have a number of goblet cells showing active irritation. This specimen is from an antrum.

This slide illustrates another further developed polyp showing the perivascular reaction at this point, and also the cellular infiltration of the tissue. The fluid gets into the tissue spaces and distends it, and is responsible for the pressure. The previous slides explain what I term a hypertrophic type of reaction, where there is a turgescence of the membrane. This is the result of phlebitis and lymphangitis and you have a hypertrophic type of sinusitis. That is the easy kind to diagnose. Most anyone can examine the nose, with the x-ray or clinically, and tell whether there is a polyp or not.

Here is still another type of sinusitis that I think is more easily overlooked, one where there is not a mass production of pathology. The pathology is of a different type, where the membrane may be thinner than ordinarily, and that occurs where there is a marked fibrosis of the tissue. This type usually results from an arteriofibrosis, an arteritis. Here is shown a small artery, and here is another, that shows a periarteritis and endarteritis.

Here is a vessel that is completely sclerosed. Where you have an obstruction of the arterioles the blood supply to the part is less

and naturally you find instead of turgescence, a shrinkage of the tissue, and ultimately note a replacement of the normal by fibrous tissue.

This area shows another vessel with similar pathology. To me these arterial changes progress and produce a definite pattern of sclerotic or fibrotic sinusitis. This is about what occurs in ozena, with the atrophy of the glands, dilatation of the ducts, and numerous small arteriosclerotic vessels. Naturally, the blood supply to this part is lessened and you find shrinkage of the tissue and loss of secretion, and this I refer to as an atrophic or fibrotic type of membrane.

This slide shows higher magnification of a large blood vessel showing occlusion of the lumen. In time there will be some absorption of the bone. Note the giant cells or osteoclast eating away small bits of bony material and producing a thinning of the bone, or osteomalacia.

The next slide shows arteriofibrosis with increase in the connective tissue. There is no edema and the membrane may be normal or thinner than normal thickness. As the result of all these vascular changes, secondary changes occur. These may be in the glands or in the bones. As the result of duct obstruction cystic glands occur, as is shown in this area. If they become infected abscesses develop beneath the surface mucous membrane. Dr. Macmillan showed you a cyst of the antrum. This is probably an abscess, the basis of which was a cyst of the antrum, that became infected.

In this case a larger cyst is shown in the antrum, which may grow to enormous size. Even several cysts in an antrum may occur and the picture of a multilocular antrum results.

Here is another slide showing the perivascular reaction. The surface epithelium has abscesses in it and is changing to a more or less squamous type of epithelium, an example of metaplasia.

Here is another slide which shows marked distortion of the surface epithelium, no cilia, and beginning metaplasia.

Here is another one which demonstrates abscesses in the mucous membrane itself. These little pockets contain pus. Just imagine how difficult it would be to wash out an antrum of that kind with marked metaplasia of the epithelium and abscess formation.

Here is an antral mucosa that shows normal epithelium in one area and marked metaplasia to squamous epithelium in another area.

The pathology in the next slide has impressed me greatly. It shows that a chronicly infected antrum can produce a papillomatous condition of the antrum. A papillary disease of the antrum. We have had some eight of those cases. They show abscesses in the epithelium. I think we are entitled to give this a new classification and I call it a chronic papillomatous sinusitis.

This is due to an infection, probably, of the fibrous tissue. If removed surgically it clears up. When first seen I made the error of calling them suspiciously malignant but after careful study and further experience I am quite convinced that they are not of a neoplastic nature.

This is a higher magnification showing an abscess in the surface epithelium. It would be very difficult to wash out by irrigation any such a mass.

Here is another papillary antrum showing marked metaplasia of the surface epithelium.

This is a higher magnification of the same thing: metaplasia of the surface epithelium with papillomatous change.

The next slides represent bony changes. These show the osteoclast which absorb the bone. Instead of laying down bone as osteoblasts do, they absorb bone. Osteoclasts make the bone porous and easily broken down.

This slide shows another bony process. Here is a spicule of bone with absorption on one side by osteoclasts and on the other side the osteoblasts are laying down spicules of bone. Excessive osteoblastic function results in increase in the amount of bone, so called osteitic thickening.

Here are some serial sections of the various sinuses. In this manner the tissues can be correlated and we can follow them through, from one section to another, just as we are doing in examination of the petrous bone. The ethmoids are taken out en masse with the sphenoid. The whole mass is decalcified, stained and studies made of the serial sections. This should add a great deal to our understanding of the pathology of sinusitis.

I am struck in studying the serial section of the sinuses, at the amount of vascularity in the mucosa of the nose.

DR. KERN: I would like to ask your opinion of the significance of the eosinophils in the pathological material that you examined.

DR. EGGSTON: I think that is a very pertinent question at this time, because you hear it so often said that if you find eosinophils, it means allergy. It may not mean allergy, because any chronic inflammatory process will ultimately show a proportionate number of eosinophils. I think some men have over-emphasized eosinophils in the examination of the secretions from the nose. A certain number of eosinophils are always present and particularly if there is a chronic infection. I think we have gone a little bit astray in our enthusiasm about allergy.

DR. KERN: I think the alleged significance of the eosinophils in the tissues and secretions has been unduly overemphasized. Dr. Schenck and I have been impressed with the fact that most secretions and tissue removed from allergics will at times show many eosinophils and at other times show none at all, just as circulating blood may at times have a high eosinophilia and at other times none at all, depending on the circumstances, whether they were recently exposed to that to which they are sensitive. We also agree with you that the down stroke of any leukocytosis at the subsidence of infection shows that very definitely.

DR. SCHENCK: I would like to know whether you noticed in this section whether thrombosis was more prone to occur in the allergic specimens or those specimens probably largely due to infection.

DR. EGGSTON: In allergy you probably do not find thrombi unless there is also a secondary infection. The thrombi are difficult to demonstrate unless the section happens to be taken in the right area. However, if serial sections are studied one is more likely to find the thrombus vessels.

CHAIRMAN SEYDELL: In some of the patients who come to us with this self-made diagnosis of sinus infection because of discharge or for some other reason, we know that they are suffering with something else, one of the variables, be it allergy, vasomotor trouble, or deficiencies. This part of the subject will now be discussed by Dr. Harry Schenck of Philadelphia.

Panel Discussion

SINUSITIS

* * *

Allergy, Vasomotor, Deficiencies

H. P. SCHENCK, M.D.
PHILADELPHIA

Allergy:

INFECTION alone, allergy alone, or both infection and allergy may produce sinus disease. This discussion will be confined to sinus disease resulting from both infection and allergy because this type of sinus disease presents many vexing and unsolved problems. My discussion will solve none of these problems. It merely presents certain clinical observations, and my interpretation of these may differ from yours.

Involvement of the sinus mucosa is the rule in patients with seasonal hay fever or pollen asthma. If,[1] however, these patients are examined prior to, or shortly after, the pollen season, the incidence of sinus abnormality is found to be no greater than that in a control group. This indicates that the mucosal swelling subsides in the absence of complicating infections, since pollinosis occurs at a time of year when infections of the upper respiratory tract are at a minimum.

On the other hand, patients with perennial nasal symptoms, of obvious allergic origin, present an extremely high incidence of sinus abnormality.[2] Chronic sinus disease has resulted from a combination of allergic changes in the sinus mucosa and infection. Prolonged exposure to allergens produces edema of both nasal and sinus mucosa. Mucosal edema is followed by closure of sinus ostia and partial absorption of air in the occluded sinuses. Stagnation of the profuse secretions of allergy occurs in the nasal chambers because physiological drainage is impossible in the face

of occlusion of the airways by edematous turbinates and impairment of ciliary action. Thus, the allergic state, continued into those seasons of the year, when infections of the upper respiratory tract are ubiquitous, has prepared a fertile field for the reception of micro-organisms. While[3] the allergic individual is apt to be less susceptible to infections in general than his non-allergic neighbor, nevertheless, tissues sufficiently altered by allergic phenomena are especially prone to infection. Thus,[4] the mucosa of both the upper and lower respiratory tracts invariably becomes infected if the allergic manifestations of those areas continue indefinitely.

Extension of infection, from the nasal passages to the interior of the sinuses, would seem to be prevented by the occlusion of the ostia by mucosal edema. This barrier may be effective for a time but the lowered tension of the air within the sinus facilitates aspiration of infected mucus whenever the edema about the ostia momentarily abates. Contiguous extension of infection along the mucous surfaces also occurs. Upon reaching the sinus mucosa, regardless of the mechanism of its approach the infection finds a soil prepared for its reception and perpetuation. The mucosa, altered by allergy, has lowered tissue resistance, ciliary action partially or completely lost, retained secretion, and ostia mechanically imperfect. Thus initial acute infection readily becomes chronic.[4]

Chronic infection, established within an allergic sinus, introduces another factor — that of bacterial sensitization.[5] There is considerable clinical evidence that bacterial sensitization follows continuous exposure to antigenic substances arising from infection in the body, but some allergists remain skeptical of the role of bacterial allergy. Their skepticism is partly based upon the inability to produce an immediate skin reaction to bacterial protein, and in part to the disappointing results of vaccine therapy or surgical removal of suspected foci of infection. The riddle is further complicated by the fact that infection lowers the threshold of absorption for antigens.[5] Exposure to a given amount of allergen produces more intense allergic reaction in the presence of infection in the shock organ than in the absence of infection in that organ. Thus, with a given pollen concentration, a hay fever patient with nasal infection will react more violently than if infection is absent. It is probably superfluous to note here that factors other than infection lower the threshold of absorption to

allergens, notably septal abnormalities producing lateral contacts. In the light of our present knowledge, it seems expedient to hold to the concept of bacterial sensitization.

While it is true that in an allergic individual, sinus disease usually develops secondarily to the allergy, there are instances in which infection appears to precede manifestations of allergy. Infection, followed by mucosal edema, in a patient of allergic heredity, may be followed by true bacterial hypersensitiveness in a minority of instances. Moreover, infections in the upper respiratory track of an individual with a familial history of allergy, even though he may have failed as yet to manifest any symptom of allergy, render the mucosa more vulnerable to allergens and pave the way for sensitization to inhalants.

The possibility of sensitizing a patient of allergic heredity by operative measures, undertaken during an active pollen season, deserves reemphasis.[6] At such seasons, surgery, other than of an emergency nature, is not justified, not only because of the possibility of sensitization but also because of possible local postoperative complications.

The allergist in the management of a patient with food allergy may so restrict the diet that serious discrepancies in nutrition ensue. Since undernutrition is an important factor in the etiology of sinus disease, as well as infections in general, drastic nutritional restriction in an allergic patient may be the precipitating factor in the production of sinus infection unless certain protective agents such as proteins, minerals and vitamins are supplied during the period of strict dietary restriction.

There has long been controversy as to whether early or late surgery should be attempted upon sinuses involved by both infection and allergy. Some clinicians have even denied the desirability of any surgical intervention. It is true that occasionally the spontaneous drainage and cure of sinus infection follow the avoidance of, or desensitization to, those substances to which the patient is sensitive. For this reason alone, adequate allergic investigation and treatment should precede any operative approach upon the sinuses unless, as may occasionally occur, the infection tends to become systemic or threatens contiguous structures. In many instances, however, neither the allergy nor the infection can be controlled in the face of insurmountable mechanical difficulties due to thickened mucosa or anatomical variations because these

perpetuate the infecton and facilitate new sensitization. Under such circumstances, surgical intervention is not debatable; it is imperative.

Vasomotor:

Vasomotor phenomena are a conspicuous feature of allergic reactions but vasomotor abnormalities occur in other conditions than allergy. The term "vasomotor," applied to certain derangements of the nose and sinuses, has been so loosely applied, that various authors have, in the past, included under this heading seasonal allergic rhinitis, perennial allergic rhinitis with positive skin tests, perennial rhinitis with negative skin tests or, in fact, any abnormality of the upper respiratory tract accompanied by edema, hypersecretion and unusual instability of the turbinal tissues.

Many of these conditions were eventually shown to be of allergic etiology and to respond to allergy measures. Perennial allergic rhinitis, with negative skin tests, however, continue to be regarded as a purely vasomotor phenomenon until refinements in the technique of testing allergens gradually reduced the number of cases to which the term "vasomotor rhinitis" could be applied.

Time does not permit the consideration of the complicated mechanism designed to distribute to the various organs and tissues, amounts of blood appropriate to their relative activities. Two agents, however, are believed to precipitate alterations in the circulation. One agent, chemical substances or hormones present in the blood, is of much less importance than the second agent, the nervous system. This nerve control is partly exercised by action through the vagus and sympathetic nerves upon the heart, and partly, by action of so-called vasoconstrictor and vasodilator nerves, upon the blood vessels. The activity of the nerve centers, from which the cardiac and vasomotor impulses are discharged, is controlled by afferent impulses, coming from the various regions of the body.[7,8,9,10,11]

Vasodilation and vasoconstriction greatly alter both the thickness and the color of tissues. When vasodilation and vasoconstriction pass the limits of physiological action by excessive or prolonged activity in one direction, the prolonged hyperemia tends to produce hyperplastic changes, while prolonged ischemia tends to produce atrophic changes. If the ischemic or hyperemic states are not too prolonged, the tissue changes are reversible, but sufficiently

prolonged, the tissue changes are irreversible. Such irreversible tissue changes are characterized by altered immunological response, cytological changes, a disproportion between the cells and the intercellular fluids, abnormal metabolic activity and altered function.

Certain non-allergic manifestations, in the nose and sinuses, which seem to be due to abnormal vasomotor function, may properly be termed vasomotor rhinitis or vasomotor sinusitis. Common factors which influence the vasomotor mechanism have long been recognized. The effects of temperature barometric pressure, humidity, diet (proteins, vitamins and salts), personal habits, and diseases such as myocardial degeneration, nephrosis and endocrine deficiencies seem to be unquestioned factors. Rest and relaxation alone may at times have an alleviating effect. The multiplicity of these precipitating factors indicates that the underlying cause is not well understood, excepting in a few instances where unilateral effects are due to a strategically placed neoplasm.

In the hyperplastic cases, the symptoms resemble those of perennial allergic rhinitis, with the exception that sneezing and itching of the mucous membrane may be absent. Unlike patients with true perennial allergic rhinitis, these patients have, or have had, no other manifestation of allergy such as asthma, urticaria or eczema. The familial history is negative for allergy and skin tests fail to reveal offending allergens. Eosinophilia is neither noted in the blood nor in the nasal secretions. The main cytological change in the nasal secretions concerns the lymphocyte which appears in increased numbers.

While the mucosal changes may at first be unilateral, they soon become and remain bilateral. The mucosa is boggy and edematous but rarely assumes the extreme pallor of allergy, tending rather to maintain a granular and pinkish surface. Changes in the sinus mucosa are evident by transillumination and in roentgenograms. The serous discharge may be exceedingly profuse. When secondary infection occurs, the primary disease is often completely obscured and remains unrecognized. Even in the absence of complications, nasal occlusion eventually becomes complete.

The ischemic cases are rare but it is possible that this state may precede the condition which we recognize as atrophic rhinitis.

Deficiencies:

It is necessary for us to take cognizance of the importance of certain deficiencies in relation to sinus disease even if only to analyze the rapidly growing literature on this subject. We must decide whether the present interest in vitamins and endocrines is much ado about nothing or whether a valuable contribution to our armamentarium in combating infection and abnormal tissue changes is being made. The volume of the Quarterly Cumulative Index, January to June, 1938, contains eleven pages solely devoted to literature of vitamins — over 800 articles. While the application of this newly gained knowledge must largely be left to the clinician interested in this special field, only the rhinologist is sufficiently experienced to interpret the effects of deficiencies and their possible alleviation as far as the sinuses and upper respiratory tract are concerned. We must also remember that much of this new literature deals with animal experiments and that application to human needs requires clarification.

There is, of course, no doubt that undernutrition is just as important a factor in the etiology of sinus disease as in infections in general. We are interested in learning just what factors are concerned in undernutrition. First and foremost, the tissues of the body require a certain minimal caloric intake to remain normal and to withstand infection. Although the caloric intake may be adequate, a certain relationship between the amounts of protein, carbohydrate and fat must be observed since little if any protein is stored in the body and an irreducible minimum of protein must be supplied to maintain good health of the tissues. Moreover, a certain relationship must be observed between the amount of fat and the amount of carbohydrate burned in order to prevent incomplete combustion of fats and resulting production of ketone bodies and organic acid acidosis.

Even though sufficient fat, carbohydrate and protein are supplied in the diet, the health of the tissues may still not be maintained. While protein is a well recognized protective factor, vitamins and minerals are equally important factors.

Nine compounds having the properties of vitamins have been isolated and four of these have been synthesized.[12] Some of these compounds have been further subdivided, so that at the present time one of them, vitamin B, is believed to include at least nine specific principles. The rhinologist may well stand agast as he

surveys this array of dietary specters. Let us consider a few of the known vitamins.

Vitamin A. — Although all vertebrates require vitamin A for the maintenance of biochemical activity,[13] no evidence exists to show that the administration of vitamin A, in excess of that contained in a properly selected diet, has any specific protective action against colds, influenza or in fact, any of the common types of infection. Quantitatively, the requirement for vitamin A is small, a few milligrams daily being adequate for the largest mammals.[14] On the other hand, foods rich in vitamin A are not the least expensive of the ordinary food items.[15] Therefore milk, butter, eggs and green leafy vegetables are apt to be scarce in the dietary of those in the lower income brackets.

Vitamin A deficiency results in xeropthalmia, and nyctalopia, or night blindness, is an early symptom of such deficiency.[12] During periods of growth and pregnancy relatively larger amounts of vitamin A are required.

One group of investigators[16] found that the health of a group of infants, receiving one-fourth the amount of vitamin A present in the average diet, was just as good as the health of another group of infants receiving from 17 to 34 times as many units of vitamin A. The low vitamin A diet was, therefore, above the minimum requirement, as judged by the nutritional state of the infants and their resistance to infections. Thus, the average diet would seem to contain at least four times as many vitamin units as the minimum requirement.

We cannot entirely dismiss the problem of vitamin A deficiency, as far as the sinuses are concerned because the specific pathologic changes of such deficiency occur in many epithelial structures. Briefly, these alterations consist in atrophy of the epithelium, reparative proliferation, and differentiation of the new product into a stratified keratinizing epithelium.[13] Scattered areas of new cells appear in the respiratory mucosa, where locally distributed basal cells alone have proliferative power.[17] Continued growth and coalescence of the new cells undermines and replaces the original epithelium with a stratifying, keratinizing epithelium. In man, the keratinized epithelial cells accumulate in the glands and their ducts, resulting in the formation of cysts filled with cheesy masses of keratinized cells.[13] In human infants[17] such keratinizing metaplasia has been found in the mucosa of the nares

and maxillary sinuses. Since keratinizing metaplasia occurs in all areas of the respiratory mucosa in severe vitamin A deficiency, characteristic changes must be expected in the mucosa of the nares, the accessory sinuses and the eustachian tube.

Repair of these epithelial alterations is extremely rapid when vitamin A is restored to the diet. In rats, reparative changes in the metaplastic epithelium begin by the fifth day.[18] Separation of the superficial keratinized cells and vacuolation of the cells of the intermediate layers is followed by differentiation of cells of the deep zone (corresponding to the stratum germinativum of epidermis) into the type of epithelium originally present.[7]

Components of the vitamin B complex. — B_1 or thiamin chloride prevents beriberi in man and polyneuritis in animals.[19] It may be of value in correcting or preventing anorexia of dietary origin and in securing optimal growth of infants and children.[12] There is, however, no justification for the indiscriminate treatment of anorexia with vitamin B, and although retardination of growth is found in vitamin B_1 deficiency, other causes are to be sought in the case of most underdeveloped children.[20]

Vitamin C: cevitamic acid. — There is no doubt of the value of vitamin C in the correction and prevention of scurvy, but there is no evidence to support an anti-infective effect.[12] There has not even been established a therapeutic effect for vitamin C in the secondary infections of scurvy.[12] It has, however, been shown that individuals with chronic infection require more vitamin C than normal individuals to increase the vitamin content of the blood. Increase in the blood vitamin is reputed to have a beneficial effect upon the nasal mucosa but here again proof is lacking.

Vitamin D. — Of the ten known forms of vitamin D, only 2 have any practical value.[21] Vitamin D is a specific for infantile rickets, spasmophilia and osteomalacia.[12] Animal experiments have shown that correction of an inadequate intake of vitamin D overcomes the improper ratio of calcium and phosphorus in the diet, and provides a more economical utilization of calcium and phosphorus.[12] The requirements of vitamin D are greatest during infancy. Rickets have been held responsible for asymmetric development of the skull and the resultant impaired development of the sinuses in children.[22]

Since evidence of acute vitamin deficiency occurs only in infants, and this vitamin is not found in appreciable quantities in most ordinary foods, we may conclude that the vitamin D requirement for man is very low or usually provided for by exposure to sunshine.[23]

Vitamin E. — This vitamin appears to be a remedy for sterility in some animals but a definite proof of its value as a remedy for sterility in man has not been submitted.[24]

Less well known vitamins. — Nicotinic acid is recognized as an anti-pellagric factor and useful for patients allergic to yeast. Numerous other factors such as Factor H, vitamin I vitamin K, and vitamin P have been shown to have various importance in the nutrition and growth of bacteria, insects and lower animals.[25] No application of these to the problems of nutrition in man can be made at present.

Endocrines. — The endocrine deficiencies are not well understood but the relation of hypothyroidism to allergy and to edema is generally recognized. Allergy is rare in those with hyperthyroidism, common in those with hypothyroidism. The characteristic bogginess of the tissues in hypothyroidism provides a mechanical factor which handicaps the sinuses in their resistance to infection. Low grade hypothroidism has long been a reputed cause of chronic sinusitis because, associated with intestinal stasis, there is pallor of the nasal mucosa and a thin white infiltrate along the nasopharynx.[26]

Hypogonadism has been held responsible for frequent head colds, occurring regularly, just before the menstrual period. The symptoms often simulate attacks of acute sinusitis or true allergic rhinitis but abate suddenly upon cessation of menstruation.[27]

Experimentally, suppurative sinusitis in rabbits has been cured by the administration of cortin, but no adequate evidence has been presented to show that cortin has any therapeutic value in human sinus infections.[28] The pituitary gland appears to play a role in sinus infections only secondarily and then only after initiating general changes which are quite obvious.

The role of liver extracts and similar substances must be mentioned because of their value in maintaining blood function rather than for any specific local effects upon the sinuses.

Minerals. — Iron is of importance for the maintenance of efficient metabolism. Its presence in the average diet needs no elaboration.

A minimum daily intake of 0.45 gram of calcium is the requirement of an adult and it has been shown that the average American diet contains considerably more calcium than is actually required.[29] When dietary deficiency is found, however, the deficiency in calcium occurs 6 times more frequently than deficiency in protein.[29] Calcium appears to lessen the irritability of the vegetative nervous system and causes a constricting effect on the arterioles, thus restricting inflammatory processes. The same action accounts for its therapeutic use in the control of the edemas of allergy.

The reciprocal relation of sodium and calcium in the body is well established. Restriction of sodium chloride in the diet is regarded as facilitating calcium absorption.[30] The dehydrating effect of restricting the sodium intake is believed to be of value in diminishing the edema of acute congestive reactions.[31]

What may we conclude concerning the relation of deficiencies to sinus disease in the light of our present knowledge?

1. The average American diet is adequate as to caloric, vitamin and mineral content.
2. Undernutrition is no more a factor in the etiology of sinus disease than in the etiology of infections in general.
3. Actual vitamin deficiencies result in definite disease entities such as night blindness, beriberi, scurvy infantile rickets, spasmophilia or osteomalacia, and thus far no specificity of vitamins in the etiology or cure of sinus disease has been demonstrated.
4. When deficiencies exist, the rhinologist is no more equipped to cope with the problem without expert assistance, than he is in coping with allergy without the assistance of the allergist.
5. Infections do not tend to follow the lack of any one dietary fraction but rather tend to follow the lack of any essential factor in the diet.
6. The most common inadequacies appear to be vitamins B and C, but A, D and G are occasionally of importance, and calcium, iron and protein must be supplied in adequate amounts.
7. Vitamins B and C, minerals and protein, appear to be protective factors, since their absence from the diet in adequate

amounts results in lowering the efficiency of body defenses. The lack of fat and uncooked vegetables in the diet of children results in serious deficiency.

8. While the maintenance of an adequate caloric level is of importance in allergic individuals, too sharply restricted by a diet based upon skin tests alone, the diet must also be properly balanced and adequate as to mineral and vitamin content.

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Panel Discussion

SINUSITIS

* * *

Physiology — Therapy

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A GREAT DEAL of work has been done of a most excellent nature and of a fascinating type on the epithelium lining the sinuses, together with the activity of the cilia, and as a result of this effort our attention has been more or less diverted from tissue which is equally, if not of greater importance, than the epithelium. I refer to the connective tissue between the basement membrane, the lamina propria.

This loosely arranged lamina propria of the sinuses contains a mechanism which is of vital importance in the defense of the sinuses against the invasion of pathogenic organisms. This defensive mechanism consists of a type of cell which is present in fairly large numbers in the normal tissue. This cell is characterized by abundant cytoplasm, by a relatively small nucleus, and what is more interesting, by the kind of activity which it undergoes.

It is a primitive type of cell, and becomes autonomous. It is able to move about, and to mobilize in the region in which infecting organisms have penetrated this area of the epithelium. These cells bear various names and it will simplify matters if we resort to the use of a single name and call them all hystiocytes. On the basis of such work as Fenton and Larsell have done, a great deal of information is now available. I wish to return in a few moments to this, but I will leave this subject at this moment.

I shall speak about the armamentarium which is available to us in the management of the sub-acute and the chronic infections

of the sinuses and I will say immediately that that armamentarium is sharply limited. Up to a very recent date we all know that practically everything we have used in the form of drops and sprays has been suspended in some sort of an oily medium, and invariably the oily medium has been a hydrocarbon oil. We know that a hydrocarbon oil which finds its way into the lung, is a tremendously dangerous thing, but while we know that, some of us do not know what actually happens when these oils get into the lung. The picture is characteristic, it is typical. The presence of hydrocarbon oil in the lung results in the formation of an extensive fibrosis. The cells in the lung which are known as the dust cells accumulate this oil in large droplets so that cavities containing oil are present. As a result of this and the fibrosis, we have what may be called a true paraffinoma, a growing thing and a destructive thing. In the growth of it and in the extension of the paraffinoma we get necrosis. These individuals then have developed and die of a lipid pneumonia.

Much less well known is the fact that silver salts, whether they be in the actual salt solution or whether they be in the form of the colloid preparations, have a similar effect and they have a pernicious tendency when applied to the mucous membrane of the nose or the sinuses to find their way into the lung, and again result not in the formation of a growing paraffinoma, but in the formation of extensive fibrosis and necrosis of the lung.

And so we see that in the treatment of our sinus and our nasal condition, our armamentarium is really markedly limited.

DR. KERN: I would like to interject a thought at this point from the standpoint of the use of the silver preparations. Just as for thirty years or more argyria usually resulted when an internist gave silver for indigestion and the patient then continued for months afterwards to get the same prescription renewed, so more recently once a mother has put argyrol drops in the youngster's nose the first time she calls the doctor, she thereafter continues to instill argyrol drops any time in her judgment symptoms seem to call for it, and there have been reported not a few cases of argyria in children from the promiscuous intranasal use of argyrol solutions.

DR. NOVAK: And so we find ourselves faced with the problem of what may be done in the way of therapy other than surgery,

and I wish to say immediately that it must be granted that the chronic sinus undoubtedly calls for some form of radical surgery, but there are other things that may be accomplished preceding the surgery and the care of the case after the surgery has been done, and I will now try to focus your attention upon the immunologic approach to this whole matter, and since the time is so short, I will summarize as rapidly as I can.

I wish to quote Pasteur who many years ago prophetically said that the physician of the future will be an immunologist. I think that was never so true as it is at the present time, and I don't believe it has been appreciated as much as it is being appreciated at the present time.

What is this immunologic approach? I started out by saying there is a defense mechanism in the lining mucosa underneath the basement membrane. These cells are there and they have a function to perform. I also wish to say that there are means at our hand to influence and to control, to exert influence and control upon this cellular mechanism. What is that means of influence? It is in a general way by the employment of such antigens as may be applied locally and which do not have this destructive action on the lung and have the effect which Dr. Kern just mentioned of resulting in argyria and other undesirable things. There are such antigens, and whether they may be filtrates, bacterial suspensions in salt solutions, or whether they be non-specific protein, their employment is something which at least should be tried before the radical surgery is attempted.

I have several notes to comment upon other measures that are of use, and the first of these is physical therapy, and the first thing under that is heat. I am no longer convinced, although I did for a long time play with various gadgets and devices in physical therapy, that the source of heat is important. I am convinced now that the source of the heat is not of very great importance, and whether a man in his predilection will choose diathermy or short-wave diathermy or a lamp or a hot water bag, the important fact is that he will use the heat, and so you have the employment of the heat on the basis, first, of its influence upon the circulation, which you just heard about from Dr. Eggston, and secondarily the effect of the heat on the immune responses of tissue, because we know with heightened temperatures the immunologic character of tissue is enhanced.

Secondly, the use of light. There is something in that, and the living tissue exposed to fluorescent light and then exposed to a short-wave ultraviolet light, does have something happen to it. The large mass of invading organisms are destroyed, and with their destruction the tissues then are perhaps better able to cope with the situation than if that mass were not destroyed by such a means as an ultraviolet light.

One comment about nasal suction. There are those who believe, on the basis of physical facts and physical experiments, that the application of mild suction to the nose cannot produce any effect within a sinus, that inasmuch as it is a closed, solid cavity you can't suck on the opening of a closed, solid cavity and draw anything out, but as a matter of experience, not any one individual but all of us, we have all looked into a nose which we knew had attached to it an infected sinus containing fluid, and upon examination found no fluid in the nose, pus or any other kind, and by the action of suction have found immediately afterward a large amount of pus produced in the nose. I still believe, and I think most of you gentlemen agree, that mild suction, intelligently applied, is still of value in the management of acute, chronic and sub-acute sinus disease.

One comment about x-ray which already has been commented upon. I do not believe there is enough experimental evidence, either on animals or on the human, to justify anyone in deciding one way or the other whether x-ray therapy in sinus disease is desirable or not, and one must forever bear in mind that in the employment of radiation we are perilously close to an extremely delicate structure of which we know very little. I mean the pituitary gland.

As for conclusions, I will say that granted generally chronic sinus infection calls for surgery, the local measures which I have touched on briefly are worthy of trial in the conservative approach; and second, local measures are essential usually following surgery.

CHAIRMAN SEYDELL: We have decided that the patient has a chronic sinus infection. It is a problem of operation and Dr. Hill will now tell us how to evaluate the conditions as they exist, and what surgical measures should be employed.

Panel Discussion

SINUSITIS

* * *

Indications for Surgery in Sinusitis

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ASSUMING that we have already made a diagnosis of sinusitis, and have used our conscience in so doing, what might be the indications for resorting to surgery? Technique will not be discussed. It will be assumed that it will be both as correct and as adequate as is generally possible.

We will all agree that careful study, a good history and thorough examination, including Roentgenograms, are necessary before deciding upon any operative procedure. We must not operate "upon sight." We must consider the clinical pathology and its probable effect upon the patient — what Wherry so aptly called "the infection load." Similar conditions in different individuals present different problems. One person may be quite content with a sinus discharge which another could not tolerate.

If operation is decided upon, the particular type must be selected according to the demands of the individual case. We must fit the operation to the patient, not the patient to the operation. Probably in no field of surgery is careful, conscientious postoperative observation more necessary. In most surgical procedures an organ or portion of tissue, which is diseased, is removed and, after healing has taken place, there is little to worry about. In sinus surgery, however, we do not remove the sinuses; we drain them, break down and remove cell partitions and remove diseased lining membranes. But we still have the sinuses to deal with; drained, ventilated and relined, and with the possibility of subsequent scar tissue formation, or reinfection, defeating the purpose

of our operative procedure. Consequently patients who have undergone operations on the sinuses must be kept under observation, and the physician must be prepared to deal with changing conditions, due to formation of scar tissue, or reinfection.

Unsatisfactory results, in any given case, assuming the technique to have been adequate, may be attributed to the inappropriateness of any surgical procedure, to incorrect selection of the particular type of operation, or to failure in proper post-operative care of the patient.

Any classification, beyond that of acute and chronic, is difficult, and bound to be arbitrary. Usually this is more satisfactory in print, than in the office. It is difficult to pigeon-hole patients.

Acute Sinusitis. — Most cases are self-limited and indications for surgery are rare. A conservative attitude should be maintained, unless complications ensue, or there be a delay in resolution and evidence of inadequate drainage. Continued pain, purulent discharge and the Roentgenogram may suggest such minor procedures, as infracture of the middle turbinate, turbinotomy or opening the agger nasi cell. Irrigation of the antrum through puncture, or, better still via the natural ostium, may be indicated; but this should not be continued ad infinitum ad nauseam. A large intra-nasal opening underneath the inferior turbinate is preferable to too many repeated punctures, with their continued trauma. Emphasis, however, should be on conservatism. We should endeavor not to interfere with the natural defense mechanism of the body.

Acute Sinusitis with Complications. — Occasionally we may encounter a fulminating type of sinusitis; or one in which we are faced with some such complication as orbital, or lid abscess, osteomyelitis, or even cavernous sinus thrombosis. These are usually in younger people and perhaps the worst cases following swimming. Here it may be necessary to abandon our conservative attitude, but our surgical efforts must be properly directed and sometimes restrained. We should endeavor to get adequate drainage with a minimum of bone work. With orbital abscess, external incision and drainage of the ethmoid, without any attempt to exenterate the cells, is preferable in the acute stage. Later, if resolution has not taken place an ethmoidectomy, intra-nasal, or external, depending upon the conditions, may be performed. In the

presence of osteomyelitis, usually diagnosed by the presence of pitting edema extending over the frontals, a radical attack without procrastination is necessary. As pointed out by Mosher, the Roentgenogram is about ten days behind time, although it may suggest potential possibilities by showing large diploic veins. Skull plate from well beyond the line of edema should be removed, and the skin flaps left open for drainage.

Acute Exacerbations of Chronic Sinusitis. If possible radical measures should be avoided, or postponed, allowing the acute process to quiet down. Again, drainage with a minimum of bone work, if interference becomes necessary.

Chronic Sinusitis — Purulent. — In maxillary sinusitis of dental origin, characterized by purulent and rather foul discharge, and dental pathology; if the condition does not resolve after removal of the dental infection and conservative treatment of the antrum, a large intra-nasal antrotomy generally suffices. In some cases there may be sufficient pathology in the floor of the antrum to warrant operation through the canine fossa. But, as a rule, the intra-nasal procedure should be tried first.

Many cases of maxillary sinusitis may have an associated ethmoiditis or the antral infection may be secondary to ethmoiditis. In certain of these, proper medical care, especially attention to endocrine dyscrasias, may prove very beneficial. In children, especially, correction of dietary errors and removal of tonsils and adenoids will frequently be followed by resolution of the sinusitis. Headache, discharge, nasal obstruction, laryngeal or pulmonary symptoms or evidence of focal infection may, however, indicate the advisability of operative interference. Simpler procedures, if at all consistent with the clinical and X-ray picture, are more satisfactory from the patient's viewpoint. Radical antrum operation such as the Caldwell-Luc should be done in these cases only when there is definite evidence of marked mucosal pathology, which probably would not resolve following simple antrotomy; or after the intra-nasal procedure has proven futile. Removal of the middle turbinate may improve drainage of the ethmoid but persistence of suppuration may indicate the advisability of an intra-nasal ethmoid operation. Usually the sphenoidal sinus is also involved and is opened at the same time. There should be no hesitancy in doing secondary operations later, as scar tissue formation may

impede drainage. The frontal sinus, if involved, is usually drained by the ethmoid operation, as it has the advantage of gravity. It is rarely necessary to attempt any work on the naso-frontal duct. Should there be sufficient pathology to warrant external operation on the frontal sinus, the combined external fronto-ethmoid seems preferable to those types of operations which endeavor to enlarge the naso-frontal duct by burrs or rasps.

Chronic Sinusitis — Hyperplastic (with polypoid changes in lining membrane or polyposis). — Many of these cases are allergic. In these, indicated allergic treatment must be carried out. Excluding cases of pollenosis, surgery is justified despite the presence of allergy, only if the clinical picture, after careful study, would suggest operative interference anyway, if the case were not allergic. In other words, we are probably dealing with sinuses in which bacterial involvement has occurred secondarily to the allergic condition. Generally, in these cases a combination of allergic treatment and rational surgery gives the best results.

With marked nasal obstruction, discharge and polyposis, and Roentgen evidence of well-established mucosal changes in the antrum, and allergic or other underlying factors controlled or ruled out, radical operation, via the canine fossa, would be indicated. This may be combined with the transantral operation on the ethmoids, if these are involved. This method may not always be quite satisfactory as far as the anterior cells are concerned but it may be combined with intra-nasal attack on this region. If there is involvement of the sphenoidal sinus, it can be widely opened by removing the anterior wall at this time. The complete opening of the sphenoidal sinus by removal of its entire floor is not as easy of accomplishment as it sounds and I doubt if many of us pretend to do it, at least at all routinely.

The external fronto-ethmoidectomy should be reserved for those cases which defy simpler methods. Lynch, himself, said, "When all else fails, try this." If, however, one wishes to keep his waiting-room filled with chronics under observation the frequent employment of this type of operation is one way of accomplishing it, for they are usually with you for a long, long time. While it is undoubtedly more efficient anatomically, and safer and easier to do thoroughly than the intra-nasal ethmoid, the after-results are not always all that one could wish for. Prolapse of orbital contents may impede drainage of the frontal sinus. A com-

pletely satisfactory radical operation on the frontal sinus is something to be desired but not yet always attained; at least from the patient's viewpoint. The external fronto-ethmoid should be employed in those cases where intra-nasal or trans-antral operation has failed, when frontal sinus pathology must be removed, or when complications demand this external approach.

The particular type of operation should depend upon the conditions of the individual case. In most cases the Lynch type of operation answers very well and has the advantage of not disturbing the anterior frontal wall. In cases with very large frontal sinuses, however, the Killian operation may be more efficient. Certain cases might justify an obliterating operation, even with the resulting deformity. In the so-called conservative operations which open the sinus externally and attempt to enlarge the duct by means of burrs and rasps it is often difficult to maintain the opening. Therefore, if there be sufficient pathology to warrant external operation, or if the frontal sinus will not resolve after an intra-nasal ethmoid, the combined external fronto-ethmoid operation seems preferable; but only after due study and consideration.

DR. MACPHERSON: What have you found in this radical antrum operation is the effect on teeth.

DR. HILL: I am inclined to feel that is somewhat overrated. I think you may get from the local anesthetic a certain amount of lasting anesthesia. If we are careful not to curette the floor of the antrum (getting generally into technic), I personally don't feel very much concerned about it.

DR. MACPHERSON: You don't think cutting the nerves has any bad effect on the teeth?

DR. HILL: I think we shouldn't make too large an opening in the anterior wall.

DR. MACPHERSON: What do you find in the way of regeneration of tissue in an antrum that has been opened up?

DR. HILL: I think most frequently it does regenerate. I think Dr. Mosher's laboratory has proven that. I have had occasion myself to reopen an antrum for alveolar fistula and pathological

studies showed regeneration of the membrane progressing. This was six weeks after radical operation.

CHAIRMAN SEYDELL: Before we take up the next item, Dr. Novak has a question he will answer.

DR. NOVAK: I have a criticism by Dr. Bricker to this effect: "I am afraid I cannot agree with Dr. Novak that diathermy is not efficacious."

There is no point of disagreement as far as I can see. My point was that the employment of heat is the important thing. The source of the heat and the manner in which it is obtained are of secondary importance and of technical interest only, perhaps. I personally do use diathermy at times. Sometimes I am perfectly content to use a hot water bag or a hot light.

In this matter, the efficiency of a diathermy machine is a great help. You get a penetration of heat, perhaps, more readily, more completely, perhaps more efficiently than you do with other sources of heat, but the important thing is the heat and not the source or the manner in which it is generated or from which it is obtained.

I think it is more important to understand the mechanism of the reaction of the tissues and the types of cells that are involved than it is to consider and pay too much attention to the manner and the way in which the heat is brought to or developed within the tissue.

DR. MACPHERSON: Dr. Novak, do you use coagulation of the turbinates?

DR. NOVAK: I have used coagulation of the turbinates. It is simply another modification of cauterization.

CHAIRMAN SEYDELL: Many of the cases that come to us with sinus trouble or vasomotor trouble or allergy, or other conditions, have systemic symptoms. Dr. Richard Kern of Philadelphia will now discuss General Medicine or the Internist's View of Sinusitis.

Panel Discussion

SINUSITIS

* * *

The Viewpoint of the Internist*

RICHARD A. KERN, M.D.
BY INVITATION
PHILADELPHIA

WHEN an internist airs his views on sinusitis before a group of rhinologists it is largely a matter of carrying coals to Newcastle. Yet he is not wholly without justification in so doing, not merely because he is usually the first to see these patients, but because in the majority of instances, sinusitis, far from being a simple local nasal problem, is intimately connected with disease elsewhere in the patient. To call attention to some of these relationships of sinusitis to extranasal troubles is therefore the chief purpose of this presentation.

Acute Sinusitis. — Acute sinusitis occurs as frequently as the common cold, and in most cases is probably as transient. Only rarely, when there is the unfortunate combination of secondary invasion by a highly virulent organism and a patient with little or no resistance, does there develop the surgical emergency of an acute fulminating sinusitis which is well known to you. Far oftener does a repeatedly occurring acute sinusitis pave the way for the establishment of a chronic involvement.

There is one complication of acute sinusitis that deserves emphasis. Under the name bronchosinusitis, Wasson[1] called attention to a condition which invariably is first seen by the internist. The patient is usually an adolescent or young adult, whose illness begins with coryza, cough, fever and the usual manifestations of grippe. But instead of clearing up in a few days, the symptoms

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continue, with cough and purulent sputum, afternoon rise of temperature, and malaise, and there is a moderate leukocytosis. Physical examination reveals persistent rales in one lower lung lobe and the roentgenogram shows increased hilum shadows, with perhaps a patchy consolidation about the large bronchi. Everything seems to point to a primary lung involvement, and often tuberculosis is suspected. Yet the underlying trouble is a silent purulent sinusitis, the relief of which is followed by prompt clearing of the lung lesion. If the sinusitis is not discovered, the condition may continue for months or years and lead eventually to serious structural damage, such as a localized bronchiectasis.

Chronic Sinusitis. — Chronic sinusitis is exceedingly common in the experience of the internist. A few years ago Schenck and I[2] studied a group of apparently normal individuals who gave no history of recent respiratory disease, no history of recurrent colds and no history of obvious sinus disease in the past. Yet in 72 per cent there was x-ray evidence of sinus abnormality and 26 per cent gave positive clinical findings: in only 22 per cent were both examinations negative. This does not mean that 78 per cent of Philadelphians have active sinus disease, but it is probable that 78 per cent of them have or have had at some time in their lives a sinus involvement sufficiently severe to leave behind the scars of its ravages.

These figures for sinusitis incidence of course do not apply to all parts of the country. This was brought home to me when I compared notes with colleagues from the South and the Southwest who see sinusitis far less frequently. But it is safe to say that the Atlantic coastal region, from Norfolk to Boston and fully up to 200 miles inland, shares with Philadelphia the distinction of being a rhinologist's paradise.

Chronic sinusitis, at first at least, may be a purely local matter, as for example when an antrum becomes involved by way of a tooth lesion. Much oftener, however, it is associated with disease elsewhere in the body: disease to which it may be secondary, or disease of which it may be the cause. And it is the extranasal difficulty which commonly first brings the patient to the internist.

Sinusitis and Focal Infection. — That chronic sinusitis may act as a focus from which infection may metastasize to other structures is well recognized. Iritis, choroiditis and keratitis, subacute and

chronic arthritis, neuritis, nephritis and myositis can often be traced to sinus infection. The internist in studying such cases therefore routinely includes the sinuses among the structures to be investigated as a possible infecting focus. It is, however, a definite clinical impression that such metastatic infection is distinctly less frequent from sinusitis than from diseased teeth, tonsils or prostate. This may be due to the fact that the sinuses with their rigid bony walls are not subjected to the frequent external pressure which literally squeezes the bacteria from tooth sockets or tonsils into the afferent lymphatics and blood vessels.

Sinusitis and Infection of the Lower Respiratory Tract. — It is in relation to infection elsewhere in the respiratory tract that chronic sinusitis assumes its greatest importance. If there is one thought above all others that I would like to leave with you, it is this: We must always consider the respiratory tract as a whole: an infection of any part of it can, by direct extension along the mucosa, involve any other part. If the infection be chronic, then the chances of its extension from one part of the respiratory tract to another are all the greater. Since the sinuses for anatomical reasons are peculiarly liable to become the site of a chronic infecton, it follows, not only that chronic sinusitis is very common but also that chronic sinusitis is both the commonest cause and the commonest complication of other chronic respiratory infections.

Chronic sinusitis is a common cause of infection lower in the respiratory tract. From time to time intercurrent factors such as exposure, fatigue and illness by lowering the patient's resistance, permit the sinus disease to flare up. Purulent discharge repeatedly infects the nasopharynx, trachea and bronchi and so a chronic bronchitis can arise. This in turn may lead to structural change, notably bronchiectasis, which makes the continuation of bronchial infection a permanent affair. Thus it is highly significant that bronchiectasis is not only invariably accompanied by chronic sinusitis, but the sinus involvement is so extensive in most cases as to be described as a pansinusitis.[3] An important clinical point in connection with bronchiectasis is that these patients are usually young persons, often children, in whom for years the disease causes little obvious trouble beyond a chronic cough to which both patient and family have become accustomed.

Dr. Hill: Could I inject a question here? How often do you feel that chronic sinusitis is the etiological cause in bronchiectasis?

Don't you feel there is a possibility that many of these cases are due to other respiratory infections such as pneumonia, and perhaps we have been putting a little too much emphasis on the sinuses as the 100 per cent cause of bronchiectasis?

DR. KERN: Most decidedly. I am perfectly certain I have seen cases in which whooping cough led on to chronic bronchial infection, and that in turn infected the sinuses. Once the sinuses were infected, however, then they acted as a secondary focus to keep the bronchial infection going.

Since children commonly swallow their sputum no attention is called to the purulent bronchitis and the gravity of the situation is long overlooked. Invariably in these patients the extensive sinusitis is practically without symptoms.

On the other hand, chronic sinusitis may arise from chronic infections lower in the respiratory tract. In any condition with chronic cough and purulent sputum, the constant spraying of the nasal chambers with the coughed-up pus will in the majority of instances result in the chronic infection of the sinuses. So one usually finds chronic sinusitis not only in bronchiectasis, but also in long-standing lung abscess and ulcerative pulmonary tuberculosis with mixed infection.

It is often impossible to tell in a given case whether the chronic infection was primary in the sinuses or in the tracheobronchial tree. The commoner assumption is that the sinuses were first involved. But that the sinus infection may be secondary has been proved beyond question. So we have seen patients with post-tonsillectomy lung abscess in whom the sinuses when examined within a few weeks of the development of the abscess were normal, but a month or two later were the site of extensive chronic infection.

The point of outstanding importance in these cases with both sinus and bronchial infection is that a cure cannot be effected unless both sinuses and bronchial tree receive thorough and intensive treatment. Yet in case after case all the treatment is directed against the major lesion in the lower respiratory tract, while the sinus disease is overlooked or ignored.

Fifteen per cent of all of us are definitely allergic. The respiratory tract is the commonest site of our allergic manifestations. If these allergic troubles are present in the colder seasons of the year when respiratory infection is prevalent, then the allergic edema is

a potent factor in the development of chronic sinusitis in these patients, as Dr. Schenck has pointed out. In our experience 90 per cent of patients with perennial allergic rhinitis and 86 per cent of perennial asthmatics have chronic sinus involvement.[4] The consequent implications in the study and treatment of these patients are obvious.

Diagnosis. — It is presumptious on my part to say anything about the diagnosis of chronic sinusitis to a group such as this. Nevertheless, I will hazard a few thoughts from the standpoint of the internist. He must realize, in the first place, the frequence of chronic sinusitis in the presence of any chronic respiratory infection and therefore must routinely have the sinuses investigated in such patients. In the second place, long experience has shown that neither the clinical nor the roentgenologic method of sinus examination is infallible. Each must supplement the other. Just as the first massage of the prostate may fail to express the pus which is found on later examination, so the clinical investigation of the sinuses is not necessarily ended by the first negative report, especially in low-grade chronic infections of the ethmoids. An antrum, although full of polyps, may nevertheless be clear to transillumination. On the other hand, the x-ray may occasionally miss a recent acute involvement, and a positive x-ray finding may be due to disease in the past and now no longer active: witness, for example, the permanent changes in the x-rays of sinuses that have been subjected to surgical procedure. The internist also would like to put in a plea for complete reports by the rhinological consultant on his findings in a patient. He can scarcely be satisfied by a nose and throat report in a ward case that has been boiled down to a mere "tonsils diseased; advise removal."

Prophylaxis. — While the treatment of sinusitis certainly does not fall in the sphere of the internist, he is decidedly interested in the matter of prophylaxis. Until we know how to prevent the common cold, we are of course powerless to do anything about the most important cause of sinusitis. The use of so-called stock cold vaccines has been much debated and to no satisfactory conclusion. It is my personal conviction, however, that such vaccines distinctly lessen the incidence of the purulent secondary infections in many, though by no means all patients. Of decided practical value in the prevention of sinusitis is the correction of major anatomical defects which favor sinus infection and interfere with the normal

drainage of these structures. In the allergic individual, the early recognition of the causes of the edema of his nasal mucosa will not only prevent the development of sinusitis but may often lead to the spontaneous cure of an existing infection.

But let us not forget such simple, but by no means trifling, matters as teaching our patients how properly to blow their noses. It is futile to forbid our patients to frequent that most prolific source of sinus infection, the swimming pool. Yet much could be accomplished — far more than is being done in the light of present knowledge — to make the swimming pool less dangerous. It is known that proper chlorination of the water will render it sterile. Yet some communities have no regulations governing this matter, and in most communities the existing regulations are neither adequate nor properly enforced. Pennsylvania law requires that a chlorine concentration of one-tenth part per million be maintained. Yet experience in our own community, Lower Merion Township, has shown that it takes two-tenths part per million to insure sterile cultures after 24 hours' incubation. Moreover, to keep the water up to standard, it requires frequent sampling and an occasional threat to close the pool when the analysis shows less than the two-tenths required by our ordinance. But it can be done and we feel that the incidence of sinus infection contracted in swimming pools can be distinctly lowered. Here, then, is a chance for a little missionary work in your own community.

In the meantime it will be my task to preach the doctrine of the implications of sinusitis to my internist colleagues, who though they will be much less appreciative of my efforts than you, will need it far more.

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CHAIRMAN SEYDELL: We come now to the final paper on the program. Dr. Duncan Macpherson will summarize all of the foregoing papers.

Panel Discussion

SINUSITIS

* * *

Summary

DUNCAN MACPHERSON, M.D.
NEW YORK

HISTORY TAKING — DR. RIDPATH

EVEN THOUGH specializing Dr. Ridpath does not neglect the general viewpoint. A diagnosis of appendicitis made and found by Dr. Ridpath, a rhinologist and the comment of the internist reminds me of having made a diagnosis of hemorrhoids by seeing marked dilated veins at the base of the tongue.

History taken by Dr. Ridpath: Symptoms of pain well covered.* In suspected sinus pus cases ask patient "How often do you blow your nose in an hour or a half a day?" Not "How many handkerchiefs do you use?" Paper is often used instead of handkerchiefs and thrown away. History of a cough is very important being one of the upper air tract symptoms not commonly in the minds of the internists. Post nasal dripping is a very important symptom. Vertigo is much more likely to be due to systemic than to sinus or ear trouble.

Examination should be systematic, beginning with the epipharynx before getting the patient gagging; then following a system in order to overlook no area. A garrulous patient diverts thought and where examination is not systematized some area may be overlooked.

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* However, two nurses of the same name on whom it was necessary to exenterate the ethmoid labyrinth and to open the sphenoids, regularly referred pains on probing the wide open sphenoids, over a period of months, the one definitely to the vertex, the other as definitely to the occiput.

Transillumination is of psychological value only, especially in men, where bones are heavy. In children and women with light bones it is an adjunct of some value. Antroscopy is of value in "window" cases. Radiographs, if good, may uncover disease in remote cells that a clinical examination might not reveal. The diagnosis should not be made by radiographs alone.

RADIOGRAMS — DR. MACMILLAN

I emphasize the importance of lateral views to show the teeth, thickening or feathery condition of the membrane over the septa of the ethmoids and for a lateral view of the frontals.

Films are brought in to us that are an insult to the intelligence of those who have to utilize their value — some photographed by doctors — others by technicians.

I do not believe the public gets good value by doctors supporting laboratories directed by technicians where certain interpretations are likely to be wrong.

Russell, of the Royal Society of Medicine, says he has films that show blocking of the sinus ostia from oedema as effectively as would bone and so completely fill the sinus as to exclude air.

Diagnosis should not be made by films alone. Clinical evidence is more important. Certain albuminous and gelatinous accumulations are pervious to the ray and show no shadow. Sometimes no culture of the usual organisms are present, although the gelatinous matter is cultured.

I do not feel that I can or should exclude sinus disease without the help of a radiograph and even then it is sometimes difficult.

A fair criticism is that laboratory workers, including radiographers, are too positive and indirectly we clinicians drive them to it.

PATHOLOGY — DR. EGGSTON

Dr. Eggston's discussions are always illuminating and often over my head. That hypertrophic and atrophic disease may coexist in the same sinus or the same turbinate, in the latter being visible even macroscopically, is proof of Dr. Eggston's statement of vessel disease.

Blocking of capillaries causes starvation in one case and atrophy, daming of return flow causes dilation of lymphatics and veins in another, while inflammaton with repeated attacks results in hyperplasia.

Dr. Eggston's work on optic nerve swellings is, as far as I know, the first serious contention with proof that arteritis frequently accompanies optic nerve swelling of sinus disease. Dr. Lewis Coffin some years earlier pointed out the presence of phlebitis in sinus disease, especially in children.

Lung infections are common in sinus disease, bronchiectasis being a complication. Arthritis may be present in sinus disease without local objective or subjective symptoms of sinus disease but often clears by treatment of sinuses.

For culturing it is sometimes helpful to resort to centrifuging of the fluid returned from the irrigated antra.

In chronic disease, sclerosis of the bone and some pathology is fixed. In allergy, oedema disappears leaving no anatomic sign of chronic pathology. Bilateral ethmoiditis of the same degree, so far as radiographic appearances are concerned, may be present in suppurative disease hence suppurative disease of sinuses is not excluded by a positive bilateral picture as claimed by some.

ALLERGY — DR. SCHENCK

The subject was discussed interestingly and well. But allergy is the New Deal in Medicine, sometimes referred to as a racket, fairly or unfairly. Infections due to allergic swellings interfering with drainage cannot be uncommon. Dean states that blocking of an ostium for 24 hours promotes bacterial growth — even more likely, of course, in cases where the blocking is due to bacterial infection. According to Cameron, in allergic or sensitization cases, eosinophilic infiltration and oedema are present together with plasma cells infiltration but with no tie-up to micro-oragnism. The cause is more likely metabolic poisoning. Also, all have noted the effect of exercise on nasal turbinal blocking in such cases.

Operations on suppurative and polypoid sinuses result in amelioration of asthma in a large percentage of cases, in cures in a small percentage of cases and in still others no benefit follows; but in all cases where polypi and suppuration are present the nasal disease should be treated.

Food in allergy. Skin tests are helpful in diagnosis but frequently there is no reaction even where a food is the cause. We often have to fall back on watching the effect of food. In other words, trial and error are resorted to.

For allergic patients I call for the help of the allergist even if it may be an honest racket in the hands of some.

ALLERGY — FOREIGN

It may interest Dr. Schenck to know that Denmark, last year at least, had but one case of hay fever. There is much literature on sinusitis and allergy in American journals. Apparently everybody writes in America and apparently only those who rank high as a rule write in Europe. Very little allergic work is being published there.

Germans often ignore organic causes and treat allergic condition as a psychosis. The French allude to the subject, largely through reference to asthma. Dr. Dekker, of Germany, says only psychic asthma is asthma. There is great confusion of ideas on everything connected with allergy and asthma.

In Holland only 2-3% of the population suffer from hay fever. Imported grasses from the United States into Denmark were responsible for the one case reported in Denmark of autumnal hay fever. Dr. A. Dysnich reports desensitizing to histamine with very good results. Histamine can be given subcutaneously 1 cc. of a 1/1000 solution, he states.

According to Dr. Valin, in 60 per cent of asthma cases he found nasal lesions and this apparently is the data given by many others in Europe. Many years ago I reported 3 cases of asthma following nasal operations in my own experience. Dr. Valin reports 4 more.

Some men claim as many as 75 per cent of cures by operative work — others merely say the nose cannot be entirely dismissed as a cause of asthma — get no benefit from operation. I am sure a large majority of my cases having no asthmatic manifestations show some nasal abnormality. Nasal abnormalities are so common that deductions drawn from the presence of these almost universal conditions should be made with care.

Drs. Jacquelin and Bonnet in France, using auto sero therapy, report that ten cc. of blood from a vein with serum separated from the clot — injected into the skin gave poor results. Two cc. to 5 cc. daily for 20 doses gave good results.

PHYSIOLOGY — DR. NOVAK, JR.

Local applications:

Silver salts are contraindicated to a degree. I had 6 cases of argyrosis coming to my office during one year. One is dead, five are still patients. The staining is fixed for life. Use of oil is contraindicated to a degree as emphasized in the paper. Ephedrine is

useful for promoting drainage. It loses its effect if prolonged and in some cases produces more obstruction. Many people — maybe 10-15 per cent — are allergic to adrenalin locally manifested by coryza. Infra red is of some use to relieve pain.

Dr. Alexander F. Laszlo used short wave therapy for 1½ years in a series of cases in one of my clinics, making notes on red and white cells and sedimentation, also effect on eosinophiles; he reported no beneficial effect. Displacement treatment is useful in the hands of some.

In x-ray therapy, which is being revived again, claims of benefit are made, often, I think, by drawing conclusions from impressions instead of from progress notes in each case. I feel no two cases of sinus disease require exactly the same treatment. The doctor in charge will shift treatments deciding which kind of local treatment a particular case requires.

Suction — very useful in both acute and chronic cases — drainage being required in all cases of acute and chronic sinus disease. It brings up fresh troops. Coagulation of turbinates is useful in many cases.

European work in physiology in the last few years is largely through attention to the sympathetic route, the removal of the sympathetic ganglia of guinea pigs apparently resulted in connective tissue formation. Ide, even, believing that ozena might be caused by operations on the sympathetic.

There is proof that the more virulent infections may not present severe local disturbance but may manifest themselves first at the clavicle while the upper glands are macroscopically unimpaired clinically.

In secondary sinus operative cases — frontal and maxillary — I never saw a case in which fibrous tissue did not form following the primary operation. That some ciliated epithelium may have formed may be true but the predominating, in fact all tissue was macroscopically fibrous in the cases that I saw opened at secondary operations. Packs of ichthyol and glycerin stimulate secretion and together with suction act as auto drainage carrying away infection with the discharge.

The displacement method, I and my office staff associates found difficult with Dr. Proetz' alternating suction and pressure syringe. His supine position is more easily applied and is quite generally used.

SURGERY — DR. HILL

One always agrees with Dr. Hill. Acute sinusitis, speaking in percentages, rarely needs any surgical cutting. In cases of orbital abscess due to sinus disease the external sinus operation is the preferable procedure. Orbital cellulitis with oedema of the lids may resolve by internal treatment, internal nasal surgery or external applications. Osteomyelitis I have seen do relatively well with the help of bacteriophage locally, internally and externally and by intravenous and intercellular injections.

I believe with Dr. Hill that acute exacerbations are better treated non-operatively, at least until the acute stage subsides. Like Dr. Hill, in maxillary sinusitis I first try irrigating, considering and excluding the teeth as a factor. Then resort to the window — but unlike him I aim to make the opening only large enough to keep open 2-3 months and if not then resolving satisfactorily I do a radical. In advocating the smaller window I may say that if a patient gets well without a permanent opening there is less liklihood of re-infection in that antrum. I have not felt that entering an antrum through a large opening and tearing out what diseased membrane one might, was good surgery.

Although some borderline cases will resolve I have seen too many cases even where a Caldwell-Luc opening was done, the surgeon leaving what he believed to be healthy tissue in place, later developing antral oedematous areas with polypi and suppuration. This was proved by the antroscope and by a second or even third operation. As Dr. Hill states, the thing is drainage in both acute and chronic cases with, in chronic cases, the removal of all tissue since it cannot regenerate.

Operative procedures for allergic cases is disappointing and harmful unless complicated by other pathology. Regarding frontal, ethmoidal and sphenoidal work I find myself in very close accord with Dr. Hill.

I find Watson Williams' diagnostic methods very useful in sphenoidal cases. Lynch's operation is useful when the frontal is small and can be reached in detail thereby. Trans-antral effort to cure disease of the ethmoids, sphenoids and antra was used here and in Europe many years ago following the procedure of Jansen but was discontinued by his students working here — at least by those whom I knew, specifically, Dr. Passmore Berens and Dr. Arthur Duel in New York. I personally discontinued it after operating on a few cases. Objections are bleeding during operation,

only fair visibility, unsatisfactory end results, mortality higher than by other methods.

I have personally seen no harm to teeth by the Caldwell-Luc operations in antral operations.

GENERAL MEDICINE — DR. KERN

Dr. Kern takes the sanest attitude of any internist I know but we expect sanity from the profession in Philadelphia. But up to this time the internists' view of sinusitis was, taking a cross section of them, in my opinion cockeyed. However, our own focal point is none too well established.

Weak points in the internists' attitude are: Arriving at a diagnosis without making examinations. No local examination made. Advice to patients to permit no one to operate on "your nose." That "they" always make the condition worse. That they will always need treatment if operated upon. Naming all severe and many mild attacks of nasal infection *sinusitis*. Technically correct, especially in severe case but branding the individual, especially the impressionable, in many cases with "sinus" disease for life. A refuge for the doctor because of his inability to relieve some illusive symptoms due, perhaps to a disease in the other end of his anatomy.

I believe the internist would do more for his patients if he were less dogmatic where the diagnosis is doubtful. Treatment by physio-therapy is misleading to both doctor and patient unless a correct diagnosis is made. I agree that climate, especially in children, is beneficial but in cases where low temperatures run and the internist is blocked mentally he should not fall back on the sinuses as the only etiological factor.

We continually hear that the internist should become more of a specialist but it is perhaps even more needful that the otolaryngologist should practice more general medicine. The patients would profit physically and economically. Even millionaires can find something better to do than sitting around the offices of many doctors in a day. Such treatment hurts the profession — and indirectly, the patient.

TO EPITOMIZE

Dr. Macmillan: Certain jelly albuminous-like substance permeable to the ray frequently does not culture for ordinary infec-

tions. The probability of fungus growth was shown by Dr. Holland Stevenson as an active agent in such cases a few years ago.

Dr. Novak, Jr.: Very little work has been done in Europe in the last few years. Dr. Negus and others refer to Proetz' work but do not contribute much that is original except some theories relative to asthma. I have just had the foreign literature on sinus disease for the last 5 years covered at the New York Academy of Medicine.

Dr. Eggston: Chronic and acute lung infections are a rather common complication of chronic sinus disease. Arthritis sometimes clears after treatment of sinuses even with negative washings of the antra, macroscopically, microscopically and with the nasal culturing sterile.

Dr. Kern — Internist's View: Climate is beneficial and in many cases essential, especially in children. Such treatment, generally, as one would give for types of tuberculosis, i.e., supportive, change of air, high and dry with pine surroundings preferable. In children with persisting low temperatures a diagnosis may be extremely difficult.

When dealing with sinus disease and in medicine as a whole the "Yes but" man is a very important factor and functions in the interest of the patient.

Section on Ophthalmology

The American Academy
of Ophthalmology and Otolaryngology

✦ ✦ ✦

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THE OPERATIVE TREATMENT OF RADIATION CATARACT*

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NEW YORK

AT THE Memorial Hospital for the Treatment of Cancer and Allied Diseases, the writer has had the opportunity to examine 112 eyes with radiation cataract, and to operate on 25 of these at the Eye Institute of New York City. Certain conclusions have been reached regarding the operative treatment of these cases, and it is this phase of the subject that will be presented in this paper. The clinical characteristics, the histology, and the other aspects of the subject will be given only insofar as they are related to the operative treatment. In this report an effort has been made to include only cataracts that have unmistakably resulted from radiation. The criteria have been: a sufficiently large dose of radiation reaching the eye, the appearance of cataractous changes after the usual latent period of two to three years, the location of the cataractous changes in a characteristic manner in the posterior subcapsular region, and the manner of development into maturity. All of the operative cases have been studied with the slit lamp. An important finding is an anterior subcapsular haze which is seen after the posterior subcapsular opacity has developed. This anterior subcapsular haze usually has fine striations and becomes increasingly more dense, particularly in the central portion where it may ultimately produce a well demarcated white area. These subcapsular changes referred to here are due to a proliferation of the lens epithelium which will be discussed more fully later.

Of the 25 eyes operated on, 16 had intracapsular extraction, 6 had extracapsular extraction, and 3 had linear extractions.

INTRACAPSULAR EXTRACTIONS

Technique: The usual cataract incision with conjunctival flap is made. This is enlarged a little with Steven's scissors on each side so that it extends around 180° of the limbal circumference.

* From the Eye Institute and the Memorial Hospital for the Treatment of Cancer and Allied Diseases.

A small iridectomy is performed. A double-arm atraumatic black silk suture 6 0 is placed so that it includes limbal tissue on the one side, and a substantial amount of the conjunctiva and its underlying episcleral tissue on the other side. With the Arruga forceps, a rather wide bite of the anterior lens capsule is obtained over the lower one-third. Exerting counter-pressure with the strabismus hook over the sclera near the limbus below, the zonules are ruptured over the lower part by gentle manipulation of the lens with the forceps. Pressure with the hook is partially abandoned and the dislocated lower part of the lens is pulled forward and upward until it appears in the wound. The lens is then lifted straight out, having been half tumbled. The suture is tied and the iris pillars replaced. Atropine sulfate solution 3% is instilled and a binocular dressing applied.

The 16 eyes operated on by the intracapsular method were done consecutively. The capsule did not tear in any case and the zonules ruptured about as readily as usual. Vitreous was lost in one instance. This was the 16th operation of the series and the vitreous appeared after the incision was made and before the lens was grasped with the capsule forceps. The corrected vision was ultimately 20/15 and Jaeger No. 1. The corrected vision in the other cases was 20/20 or better in nine, 20/40 or better in six, and 20/100 in one. This latter case had slight senile degenerative changes in the macula. There were no instances of secondary glaucoma following the operation. In one case, secondary glaucoma developed the fourth month after the operation from an occlusion of the central retinal vein. In only one case was a post-operative hemorrhage noted. This was a 3 mm. hyphæma on the seventh day after the operation. This was readily absorbed.

HISTOLOGIC FINDINGS

The 16 lenses removed in capsule were examined microscopically, and the histologic findings relevant to the operative treatment of these cases are given.

Under the anterior capsule the epithelial layer shows various signs of proliferation. Instead of being a layer of single cell thickness, it may be several to innumerable cells in thickness (Fig. I, a). These show all metaplastic transitions, from the normal-appearing epithelial type to the long connective tissue-like cells with a fibrous intercellular structure. Under the capsule

there may be zones of cortical liquefaction (Fig. I, b) which are rather sharply outlined from the surrounding lens substance. At the deeper margins of these areas of softening the epithelial nuclei are sometimes discernible (Fig. I, c). The epithelium in places may produce rudimentary and malformed lens fibers under the capsule. These appear as sharply demarcated vesicle-like cells, some of which show nuclei and some do not (Fig. II, a). In places the subcapsular epithelium may be missing entirely. In fully matured cataracts of longer duration, the proliferated epithelium shows a more complete metaplastic change into fibrous-

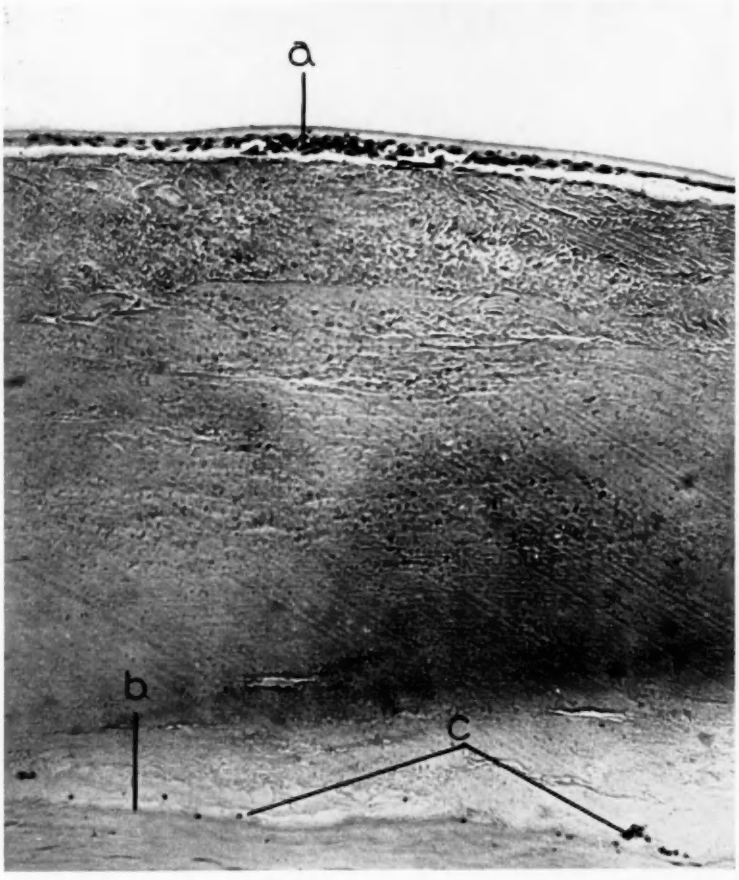

FIG. I — (Reese) — Under the anterior capsule at *a*, the epithelium has proliferated and undergone some metaplastic changes. The cortex, as far as *b* is liquified. Some proliferated epithelial cells are seen at the margin of the liquifaction at *c*.

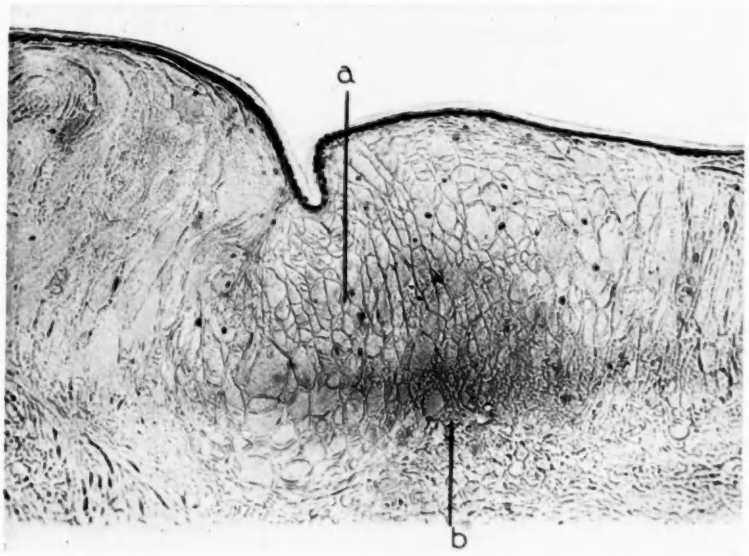

FIG. II — (Reese) — Under the anterior capsule the epithelium has proliferated into several layers thick. A layer *a* of malformed vesicle-like lens fibers, has been produced as far as *b*. The nuclei of some are shown.

like tissue under the anterior capsule, thickest in the central portion and gradually thinner toward the periphery (Fig. III, a). This fibrous plaque of tissue under the anterior capsule may be quite thick and dense and its contracture may produce folds in the overlying capsule. The epithelial cells tend to proliferate and extend also under the posterior capsule. Sometimes they are normal-looking cells, but more often they have a long nucleus re-

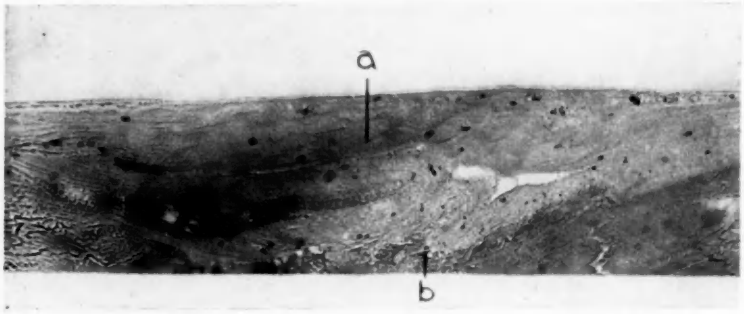

FIG. III — (Reese) — Under the anterior capsule is a layer *a* of rather dense fibrous-like tissue extending into the cortex as far as *b*. Many of the nuclei are seen.

sembling connective tissue cells and frequently these atypical epithelial cells will tend to form rudimentary and malformed lens fibers. Foci of liquefaction are also seen under the posterior capsule and atypical epithelial cells proliferate sometimes into or around it. The germinative area, or nuclear zone, at the equator of the lens, sometimes shows more, and sometimes less, active proliferation than usual. Occasionally, the nuclei are almost absent and the lens substance in this region is fragmented or liquefied. Sometimes atypical vesicle-like lens fibers are formed by these equatorial cells. Proliferation of the lens epithelium from radiation has been reported by Grzedzielski.[1]

Interpretation: The subcapsular and equatorial epithelium may receive in part a lethal dose and in part a sublethal dose of radiation. As a result of the former, the epithelium disappears, and as a result of the latter, it is stimulated to proliferate. This proliferation is an active process which not only replaces the destroyed epithelium but continues with little or no abatement. The longer the duration of the process the more the proliferated epithelial cells show the metaplastic change into the fibrous tissue and the denser and thicker this tissue becomes. This more advanced stage of proliferation and metaplasia is seen only under the anterior capsule. This older and denser fibrous tissue (Fig. III, a) may be a condensation and inspissation of the malformed, vesicle-like fibers (Fig. II, a).

One of the cases which was intended to be an extracapsular extraction will warrant some details as follows: Before the operation it was noted that an area just under the anterior surface of the lens in the central part was more opaque than the surrounding region and had a dull white color. This area, which had irregular margins, faded out gradually toward the periphery of the lens. There were three small posterior synechiae at the pupillary margin below. The cornea showed some diffuse opacities and a few blood vessels which were changes due to radiation. Because of the synechiae I elected to do an extracapsular extraction. It was impossible to cut the anterior capsule with the cystotome except in the periphery but an opening was obtained here as evidenced by the escape of some milky fluid. The anterior capsule was then grasped with the sharp-toothed forceps of Terson in an attempt to remove it. The capsule would not tear so the lens was dislocated and removed within the capsule with the

sharp-toothed forceps. The operation was without incident and the final corrected vision was 20/40. The reduced vision was due to the corneal opacity. This lens was sectioned and examined microscopically. Under the anterior capsule there was, instead of the epithelium, a layer of dense fibrous tissue six times thicker than the anterior capsule. This tissue was thickest in the central part and gradually thinned out toward the periphery so that in the region of the equator it almost disappeared. This explains: (1) the nature of the dense white area seen clinically under the anterior surface of the lens; (2) the reason it was so difficult to open the anterior capsule with the cystotome; and (3) the reason the lens could be delivered in capsule with a sharp-toothed capsule forceps without tearing the capsule.

EXTRACAPSULAR EXTRACTIONS

Technique: This is the same as for the intracapsular extraction except that a large square area of anterior capsule is incised with the cystotome and removed with the Terson forceps. The lens nucleus is then expressed and usually any soft lens matter is irrigated from the anterior chamber with normal saline solution.

In each of the six extracapsular extractions the lens nucleus was delivered without difficulty and with no loss of vitreous. In three cases the convalescence was uneventful, and the ultimate vision was 20/30 in two and 20/20 in one. In one of these three cases a discission was necessary after three months' time. Of the other two, the one died several months after the operation and the other could not be traced so the question of a secondary membrane cannot be answered. The other three eyes will warrant a more detailed report as follows:

Case I

Before the operation, the result of the examination of the eye was negative except for the radiation cataract. The intraocular tension taken with the Schiötz instrument was 17 mm. Hg. A combined extracapsular extraction was done without incident. Post-operatively, the anterior chamber was shallow for ten days. Six weeks after the operation the tension was 22 mm. Hg. Seven weeks afterwards the corrected vision was 20/20. Nine months post-operatively the tension was found to be elevated for the first time, measuring 34 mm. Hg. There had been almost a continuous slight blush to the eye about the limbal region due to a mild iridocyclitis. The tension ranged between 34 and 45 mm. Hg. even

with miotics so that a cyclodialysis operation was done two months before the writing of this paper with the result that the tension has ranged from 9 to 11 mm. Hg. since. Several months after the extraction there appeared a grayish-white tissue in the coloboma and around the pupillary area. This has become increasingly more dense but a central clear area permits good vision.

Case II

The preoperative examination of the eye was negative except for the radiation cataract. The tension was 20 mm. Hg. with the Schiötz instrument. A combined extracapsular extraction was done without incident. For ten days the anterior chamber was shallow. The eye remained slightly congested due to a mild iridocyclitis and two months after the operation the tension was 29 mm. Hg. At this time a hemorrhage occurred in the anterior chamber with no history of trauma. This absorbed within about two weeks. Five months after the operation a mild iridocyclitis was still present, and a dense grayish-white membrane filled the coloboma and pupillary area. The intraocular tension was elevated to 35 mm. Hg. Tension ranged from 35 to 45 mm. Hg. for the next month so that a cyclodialysis operation was performed. During the following three months the tension was found once as high as 40 mm. Hg. but most of the time it was normal. The grayish-white tissue occupying the coloboma and pupillary area had become so extremely dense by now that it was necessary to use a De Wecker scissors to cut an opening in it. The tension fluctuated for the following five months but usually it was elevated to around 35 mm. Hg. A slight pannus with bullous keratitis formed and only 20/200 vision could be obtained.

Case III

Before the operation some newformed blood vessels were noted in the bulbar conjunctiva and a few in the cornea which were due to radiation. A combined extracapsular extraction was done without incident. Following the operation there was continued lachrymation, pain, and conjunctival and ciliary congestion due to an iridocyclitis and secondary glaucoma. The pupillary area and coloboma gradually became occluded by a dull grayish-white opaque tissue which seemed to grow progressively more dense and vascularized. This tissue seemed to extend across the coloboma toward the site of the wound. The iris showed indistinct surface markings, many newformed blood vessels, and

around the pupillary and coloboma margins it was adherent to the newformed tissue and merged gradually into it. After eight months the eye was enucleated because of pain.

Microscopic Examination:

There is a thick, dense, fibrous membrane filling the pupillary area and operative coloboma. This membrane extends from the posterior aspect of the incision at the limbus above to the posterior surface of the iris below. It is adherent to the iris and involves the iris pillars. It is intimately associated with the remains of the lens epithelium which has been left in the Soemmering ring. There is a narrow anterior peripheral synechia and a bombé of the iris. The stroma of the iris shows in the interstices an eosin-staining fluid and a considerable number of lymphocytes which are also found to a less extent in the ciliary body. The anterior hyaloid membrane is intact. The retina, choroid and optic nerve are not changed.

Impression: The dense grayish-white tissue seen clinically in the coloboma and pupillary area represents a fibrous metaplasia of the proliferated lens epithelium. It seems probable that this is responsible for the iridocyclitis and secondary glaucoma.

SUMMARY OF EXTRACAPSULAR EXTRACTIONS

In three cases there was the usual post-operative course and ultimately normal vision was obtained.

In three cases following the operation an iridocyclitis and and secondary glaucoma occurred. In each instance this was accompanied by the formation of a dense gray to pearl-white membrane in the pupillary and coloboma area. One of these eyes was enucleated because of pain. The microscopic examination of this eye, together with the clinical course of these cases, seemed to indicate that when an extracapsular extraction is done on a radiation cataract, an iridocyclitis and secondary glaucoma tend to occur. The lens epithelium, left in the eye, has been stimulated to proliferate by the radiation and continues to do so following the operation thus producing a dense fibrous-like tissue which seems to be responsible for the iridocyclitis and secondary glaucoma.

LINEAR EXTRACTIONS

Technique: With the pupil dilated, a Ziegler knife needle is passed into the anterior chamber at the limbus under a small conjunctival flap. The anterior capsule is opened by a Y incision. After 5 to 7 days, a keratome incision is made in the cornea above, 3 mm. from the limbus. The soft lens matter is irrigated out with normal saline solution.

Of the three linear extractions, one patient was a boy 11 years of age with bilateral radiation cataracts. On the right eye a linear extraction was done and in six weeks the corrected vision was 20/20. In 2½ years, the vision had gradually gotten worse so that it was ultimately reduced to 20/200 because of a secondary membrane. A discission was done which resulted in a corrected vision of 20/20. On the left eye a linear extraction was done and in two months' time the corrected vision was 20/20. In 1½ years the vision had gradually gotten worse so that it was ultimately reduced to 20/100 because of a secondary membrane. A discission was done which resulted in a corrected vision of 20/20.

The other patient was a girl six years old who had had a bilateral retinoblastoma. The one eye had been enucleated and the fellow eye radiated. A cataract which had a dense pearly-gray color developed just under the anterior capsule. The density was greatest centrally, and diminished toward the periphery. A discission was attempted but the anterior capsule was so tough that an opening could not be made in it with a Ziegler knife-needle or a Wheeler discission knife. As the light projection was questionable and the eye soft, no further operative procedure was tried.

The dense opacity seen just under the anterior capsule was unquestionably fibrous tissue from proliferated lens epithelium and the inability to open the lens was due to this.

Proliferation of the lens epithelium is seen in some senile cataracts, and particularly those of long standing. This proliferated epithelium may also undergo a fibrous metaplasia and be seen clinically as an opaque whitish area under the anterior capsule, densest in the central part of the lens and thinning towards the equator. I believe that this epithelial proliferation, even in the less-marked instances, can be appreciated with the slit lamp as a subcapsular change. When this is present, the capsule should be sufficiently strong to withstand an intracapsular extraction. It may be these changes, as well as the frequently mentioned weakened zonules, that make lenses in elderly people suitable for intracapsular extraction.

Proliferation of the epithelium under the anterior capsule is frequently seen in complicated cataracts, and Lamb[2] has reported recently on the fibrous metaplasia of this proliferated epithelial tissue. This process may be so marked that the proliferated fibrous-like tissue protrudes quite far through the pupillary area.

The tendency for such changes in complicated cataracts explains the frequent toughness of these capsules in doing the intracapsular operation.

CONCLUSIONS

The cataracts caused by radiation tend to have a proliferation of the epithelium under the anterior capsule into a metaplastic fibrous layer. This strengthens the anterior capsule and makes this type of cataract particularly suitable for the intracapsular extraction. The extracapsular extraction in such cases is contraindicated because the lens epithelium remaining after the nucleus is extracted may continue to proliferate and form dense fibrous tissue which tends to produce an iridocyclitis and secondary glaucoma.

Some senile and some complicated cataracts also show a fibrous metaplasia of proliferated epithelium under the anterior capsule. When present this can be appreciated clinically even in the lesser degrees and is a factor in the successful removal of the lens within its capsule.

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The technical work has been done by Miss Lily Kneiske and the microphotographs by Mr. N. E. Ross.

DISCUSSION

DR. ARNOLD KNAPP, New York: I want to congratulate Dr. Reese on the admirable results he has obtained in these difficult cases. I was particularly interested in noting the adequacy of extracapsular operation in the operative treatment of radiation cataract. Unfortunately, the intra-capsular operation does not always succeed. In three of the six extra-capsular operations Dr. Reese has described, he had had a complication consisting in a dense white pupillary membrane. This he regards as due to proliferation of this capsular epithelium, which then causes the cyclitis and complications. My experience in these cases is very limited but I have been unfortunate enongh to have a case which belongs to this series of complications that Dr. Reese has described, and Dr. Reese was good enough to examine this patient with me.

In my case, the white material in the pupil was there much too early for a proliferation of the epithelium to have taken place. I therefore regard it as a retention of cortex which perhaps in these cases is particularly toxic, and which I think leads then to the cyclitis and to the dense pupillary membrane.

DR. GRADY CLAY, Atlanta: Dr. Reese has presented valuable information concerning the treatment of radiation cataract and has the opportunity to present to the ophthalmologist much greater information, namely, an estimate of the quantity of radiation that the adult lens will tolerate without production of a cataract.

Results of animal experiments have varied so much with different animals and workers and there has been so little experiment with controlled accurate dosage of radiation that such accumulated data is not altogether applicable to human eye. However, in a general sense, the lens of the infant seems to be quite susceptible to radiation, showing an early disturbance of the epithelium, while the mature lens seems relatively radio-resistant, the eyelids, conjunctivae and surrounding skin being much more sensitive.

The occurance of radiation cataract in the well organized clinic is not so frequent because the eye is generally well protected from the direct action of the rays, especially when it becomes necessary to give large dosage in the surrounding skin area, such as in the treatment of epithelioma of the lids. If x-ray or radium are carelessly applied near the eye, and radiation sequelae develop in the skin, one may expect changes in the lens leading to the formation of cataract. Yet it is surprising how much radiation an eye may tolerate without ever developing a cataract.

During the past seven years we have used x-rays in the treatment of blepharitis and infected corneal ulcers with most marvelous results. All the treatment was given in small sub-erythema doses (75 to 100 R-units) the total not exceeding one and one-half erythema doses. Corneas which appeared lost have immediately begun to heal under such radiation so that I feel that x-ray treatment is one of our most valuable forms of therapy. Recently we have examined many of these early cases with negative slit lamp findings.

The author's surgical experience has been so much greater than mine with this group that I am not in position to add much to the discussion. I have not removed such a cataract by the intracapsular method. Most of the cases I have studied developed the post subcapsular opacities resembling the so-called complicated cataract and the tetany cataract. The opacities in many radiation cataracts remain confined to the post-subcapsular area for years and for these cases I have done a needling followed by linear extraction some days later without complication. The visual results in my cases were remarkably good.

I have enjoyed reading the author's paper and am convinced that the proper method of extracting such a cataract is by the intracapsular method.

PECULIARITIES OF WELL-KNOWN EYE DISEASES IN THE DUTCH EAST INDIES

PROF. A. W. MULOCK HOUWER
BY INVITATION
BATAVIA, JAVA

I CAN'T EXPECT you to give much of your time to a discussion on eye diseases in the Dutch East Indies. The conditions over there are so different from those in your country that it may seem that they are of small interest to you. Still the problems in a territory as extensive as our colony might be worthy of your attention.

In this tropical country live 61,000,000 people, mostly natives; and two-thirds of them, 41,000,000, live on the isle of Java which is very densely populated. The number of eye patients is extremely high and so is the number of the blind. According to the census of 1930, the Dutch East Indies had nearly 140,000 blind, that is 244 per 100,000 inhabitants. This number seems to be not so very high as that number is 90, for your country. However, as the census of 1930 only those people that couldn't perceive the movement of fingers close to their eyes, were considered to be blind, we may safely estimate the total number of blind people, according to the Western definition, two or three times higher; hence we may accept that of every 100 natives more than 5 are blind. To cope with the eye diseases which cause the blindness there are 20 fully qualified eye surgeons and about 20 half-specialists; the general practitioners do also what they can to help the eye patients; their time and knowledge of eye diseases, however, is limited. You may see from this what an enormous amount of work there remains to be done in the D.E.I. to prevent and treat blindness.

But I will not trouble you any more with our worries in this respect but will tell something about the eye diseases that prevail with us. I will divide them in two classes: those that most of you don't know, as they are not met with in the U.S.A., and those that are well known to you. Perhaps you will be most interested in the first group; I think it better, however, not to speak about them. It should either take too much time or give you a quite unsatisfactory

idea of them. So I pass on to the second group. This one may be of interest to you as well known eye diseases have very often an unusual character in the tropics.

Perhaps many of you think that cataract is one of the chief causes of blindness in the D.E.I.; the astronomic figures we see when we read about cataract operations in British East India are the cause of this erroneous opinion. In D.E.I. cataract is probably not more frequent than in Europe. There are, however, a great number of cases that require operation, as in many parts of the archipelago eye surgeons are not available. The type of cataract in Java is quite distinct from that met with in Europe. Most cases, even in young people, begin as a central posterior subcapsular cortical cataract; later on comes a nuclear cataract. The perinuclear cortical cataract of the wheel-spoke-type is very rare. In my opinion the influence of infra-red rays may play some part in the cause of this type of cataract.

About the chief cause of blindness in India, trachoma, I haven't much to say. Its course and treatment is much the same as in other countries; the few improvements we have made in its treatment are of small interest to you, as trachoma is not very prevalent in your country.

Cases of acute conjunctivitis, caused by Koch-Weeks or Morax-Axenfeld's bacillus are very frequent. Especially the first kind occurs in epidemics; these are somewhat more frequent in the dry monsoon, but the differences are not very great. Many cases lead to serious ulceration of the cornea and so often to blindness. Hypovitaminosis-A is often the cause of this fatal ending.

Gonorrhoeal conjunctivitis is also very often a cause of blindness. In many parts of the archipelago gonorrhoea is very prevalent as venereal disease, mostly so in those parts close to the bigger ports and European centers, as in Java, Sumatra and Bali; in the more distant parts it is rarely met with. In my clinic I treat about 100 or 150 cases of gonorrhoeal conjunctivitis of adults yearly. In most of these cases it is distinctly of venereal origin; only in rare cases, mostly in children, the patients have no gonorrhoeal infection of the genital organs. Due to the absence of any notion of hygiene sometimes small epidemics of this disease may arise — so one of my assistants found some months ago 23 recent cases in one small village. In one case I could find the cause of a small house-epidemic. Some Chinese children and their mother

were suffering from acute conjunctivitis (probably Koch-Weeks). The mother treated these cases by washing the diseased eyes with freshly produced urine. As the mother was suffering from gonorrhoeal vulvo-vaginitis you may expect the result.

Many cases come too late to the doctor and are lost. When they come to us with a minor corneal ulceration we can almost guarantee recovery with good vision. I have no time to give you particulars of our treatment, the principal part being intravenous injection of typhoid vaccin.

Gonorrhoeal iritis does not occur in natives; in six years I saw only two cases: one in a Chinese and one in an Eurasian.

It is very remarkable that gonorrhoeal conjunctivitis in the new-born is not very frequent and in most cases benign.

From gonorrhoea I pass on to syphilis. This is very frequently seen in the same parts of India as gonorrhoea. I see many cases of eye-syphilis. During 20 years of practice in Amsterdam I saw two or three cases of gumma of the ciliary body; now I see as many cases a year; syphilitic iritis in its different forms is very frequent: I see one or two cases every month. In striking contrast herewith is the rarity of keratitis parenchymatosa of heredo-syphilitic origin: the explanation of this rarity is perhaps, that the syphilis is so serious that the children die shortly after birth and thus have no opportunity to develop keratitis parenchymatosa.

Another most remarkable fact about syphilis in India is the rarity of tabes dorsalis. Up to some few years ago tabes dorsalis was thought to be entirely absent in natives. Now we know that what was formerly diagnosed as idiopathic optic atrophy is nearly always an early stage of tabes as may be concluded from the neurological examination and especially the blood and spinal fluid test. Almost every case of tabes my colleague of the neurological department sees, has come to him from my department as in 77 per cent of the cases the first complaint of the native tabes patient is the failing of vision. Atactic cases and such with crises are very rarely seen. It is interesting to look for an explanation of this fact. One may ask, thinking of the work of Lauber and Sobanski, whether the low blood pressure of the natives may have something to do with it.

From syphilis I go on to tuberculosis. Tuberculosis in the form of tubercle of the lungs is very frequent in India, so you will be astonished to hear that tuberculous iridocyclitis and chorioiditis is

very rare; I see only a few cases every year and those are mostly rather benign. The only form of tuberculous eye disease I see very often is sclerokeratitis. Why the sclerotic and the cornea in natives are easily attacked by tuberculosis and the uveal tract so rarely, I don't know!

A remarkable coincidence is that sympathetic ophthalmia, a disease which in so many respects resembles tuberculosis of the eye is also rare in Java and rather benign. From the absence of skilled treatment of eye injuries one might expect to see many cases of this most disagreeable disease, yet they are rare.

Another disease which has points of resemblance with tuberculosis is leprosy. Leprosy is fairly abundant in D.E.I.; yet up till now I have seen only one case of keratitis and uveitis leprosa. I see rather often cases of leprosy but they come to the ophthalmological department for another reason, viz.: facial paralysis, lagophthalmus and often keratitis-lagophthalmo.

About a very interesting chapter of ophthalmology I must be almost silent. Xerophthalmia is very often seen and it ends often in kerato malacia and leads so to incurable blindness. It is even one of the principal causes of blindness in the Dutch East Indies. Our knowledge about the peculiarities of this disease is rapidly increasing, but I can't give you a good idea of it in a few words; so I had better pass on.

From the chapter of the eye tumours I want to tell you a very remarkable fact. During my stay of six years in Batavia I didn't see a single case of malignant melanoma of the uvea in a native and I couldn't find an undoubtful case in literature either. When you know that in the Western part of Java live about 11,000,000 natives and in the whole archipelago about 300,000 Europeans and Eurasians and when you hear that in the same time I saw two cases of malignant melanoma of the uveal tract in Europeans, you will come with me to the conclusion that this disease must be very much rarer in a native than in an European.

You know perhaps already that anomalies of refraction are much less frequent in natives than in Europeans; probably for this reason strabismus concomitans is rare also.

Now I have told you the most prominent differences in behaviour and frequency between the eye diseases of Europeans and the natives in the D.E.I. You will ask me the explanation of all these differences. In some cases I've said already that I don't know

them. In the case of xerophthalmia we know that the unsufficient food of the poor natives is the cause of the high frequency of this disease. In most of the other diseases we are inclined to suppose that racial, climatic and other circumstances are responsible for the differences; we must, however, be fully aware that these are not more than suppositions without a solid foundation. Geographical ophthalmology is still a very young branch of the old tree, but as you may conclude from many of the facts I've told you it promises to be a very interesting science.

OSSIFICATION OF THE CHOROID

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THE SUBJECT of intraocular ossification may be traced backward in the literature for a hundred years. Indeed, bone within the globe is a thing so apart from one's conception of the structure and function of this organ as to seem almost inexplicable. Its presence in a pathologic preparation excites curiosity akin to that caused by the discovery of a foreign body. An ordinary intraocular foreign body reveals itself by the distinct and sharply defined shadow that it casts on the photographic plate. However, the detection of bone in the choroid by this method is fraught with obstacles, on account of the shadow produced by the cranial bones. Ordinarily it is possible to obtain a "skeleton free photograph" of only the anterior half of the globe so that ossification situated in the posterior half may evade detection. In an endeavor to obtain a greater exposure, fluid may be injected into the retrobulbar region with a view of causing exophthalmos. This procedure is not justified merely to obtain a picture of a blind atrophic globe, because a certain danger accompanies every retrobulbar injection. Clinically, a roentgenogram of an inflammed blind atrophic globe is of little practical value because the indications for removal remain the same whether bone be present or not.

A very special reason why films of ossified globes so often fail to yield a satisfactory result is that it is only chalk in a bone that throws a shadow, but many times there being no chalk, the presence of bone is not detected.

A suspicion of intraocular ossification is aroused if on palpating a shrunken globe one meets with hard inelastic resistance and pressure causes slight pain. In such a case the condition is sometimes spoken of as "atrophic glaucoma." Transillumination through the sclera is confusing because one may not be able to decide whether a given shadow is produced by bone, by chalk alone as in a calcified lens, or by a pigmented sarcoma, or even by a quantity of hematogenous pigment.

It is the general opinion that atrophic ossified globes after injury are particularly prone to produce sympathetic ophthalmia, the bone being considered to have the effect of a foreign body, causing irritation. Such an eye, it is emphasized, should be enucleated.

The incentive for the preparation of this paper was the belief that an extensive and critical study of the anatomy and pathology of intraocular ossification would bring out facts of value in clinical experience and advance the understanding not only of ocular pathology but of general pathology. Most of the papers on ossification of the choroid deal with individual cases. The scope of knowledge as to the localization of bone, its method of formation and its cause and significance seems to be limited, and in some respects erroneous. Pathologic experience caused the writer to question the correctness of the commonly accepted theory that bone is a product or a mere transformation of membranes on the surface of the choroid. On the contrary, it became his conviction that this holds good in comparatively few cases.

MATERIAL

The material that forms the basis of this study consisted of microscopic preparations from 81 ossified globes, enucleated mostly because of atrophy, phthisis, or secondary glaucoma. Inasmuch as only a collection of ossified globes was studied, it was not possible to give exact data as to what percentage of all atrophic globes enucleated contain bone. In many enucleated globes the atrophy is of recent origin, enucleation having been done early for fear of sympathetic ophthalmia. One does not expect to find ossification in recent cases. A study of old atrophic globes in which injury or disease took place a number of years before enucleation would probably show that about one-third of them contain bone. However, globes that have stood in atrophy a great length of time are seldom enucleated, especially when atrophy has followed metastatic ophthalmitis or spontaneous iridocyclitis. In these latter cases the fear of sympathetic ophthalmia is not great. These eyes seldom becomed inflammed. That ossification is not rare in atrophic globes is suggested by the fact that the present survey deals with such a great number of cases.

HISTORIES — AGE AND SEX

In 70 cases notes about the age and sex were given. There were 50 males. The predominance of males was accounted for by the

fact that more males than females are exposed to injuries. According to a tabulation of the ages of 64 patients, it appeared that ossification was more or less equally distributed in the various decades. One would expect to find more cases of ossification in the later decades of life, when the body is more prone to calcification, yet there were relatively as many cases of ossification between the ages of 11 and 30 as between the ages of 30 and 60 years.

AMOUNT OF CALCIFICATION

Of 13 globes removed in the second decade of life, only 5 showed marked calcification. On the contrary, in the third decade, of 14 cases, 11 showed calcification. Of the 6 cases in the seventh decade, 5 showed marked calcification. From these statistics the conclusion was reached that advanced age is a factor in calcification. However, advanced age, as just mentioned, was not found to play a role in the production of bone. By this statement it at once becomes evident that calcification is not an essential agent in the production of bone in spite of the view that calcification is the forerunner and the chief stimulus of intraocular ossification.

DURATION OF DISEASES LEADING TO ENUCLEATION

Of the 52 cases in which the duration of a train of symptoms was recorded the longest was 55 years, in the case of a woman injured with an arrow at the age of 7 years. The eye had been painful for 17 years. Pathologically the globe was much shrunken and numerous lamellae of bone, enclosing a large quantity of fat marrow, formed a shell over the choroid at the posterior pole.

Of the cases of short duration 1 was enucleated 8 years after the onset of disease; 2, 5 years after, and 1, 2 years after. This latter presents one of the shortest durations of bone formation on record. Microscopic examination revealed a traumatic cataract, iris bombé and total detachment of the retina. Lymphocytic nodules in the iris, ciliary body and choroid indicated that the inflammation was still active. Non-calcified bone surrounded remains of the lens separated by membranes from the ciliary body. At the same time bone was present on the choroid near the papilla (Fig. 36). It is remarkable that such a large quantity of bone could have formed in so short a period. According to Parsons' *Pathology* the time required for the formation of bone is in most cases at least several years, and the record in the present paper corroborates this. Parsons mentions two cases, one reported by Schiess-

Gemuseus, in which bone was found in the choroid of a globe that had been normal 10 months before, the other reported by Leslie Buchman, in which bone was found in the choroid of a globe that had been injured 10 weeks before removal. This latter seems impossible in the light of the writer's pathologic experience, although A. Fuchs reported an analogous finding in the form of foreign body giant cells in a globe enucleated because of death four days after a cataract extraction.

GENERAL ANATOMIC CONDITION OF THE GLOBES

A majority of globes was not greatly shrunken and the sclera was as a rule not folded. Only 11 were decidedly atrophic, including a phthisic globe. From these statistics it is seen that the ossification was by no means confined to highly atrophic globes with which large amounts of membranes are associated. This fact becomes more evident when it is told that 24 of the globes were glaucomatous. It is true that in only 11 cases the presence of glaucoma was mentioned in the history, but in 13 other cases the anatomic findings made it certain that at one time the tension had been elevated.

CAUSES LEADING TO OSSIFICATION

The greatest number of causes that led to ossification given in the histories of 78 globes were traced back to perforations, 33 being of traumatic and 4 of surgical origin. In 16 cases corneal lesions had affected the interior of the globe. Endogeneous diseases such as spontaneous uveitis and metastatic end ophthalmitis accounted for 25 cases. The cases showing retinal detachment were especially interesting because apparently toxins formed locally in the subretinal fluid were a factor in the production of the bone.

DIFFERENT TYPES OF BONE

In a general review of the material it was possible to differentiate 3 types of bone formation according to shape.

First. — Plates of solid bone in various locations (Fig. 13).

Second. — Thick shells of bone, occasionally measuring as much as 7 mm. in thickness. This type had an elaborate spongioid character, possessing a number of trabeculae joined by small bridges of bone, all of which enclosed a substantia spongiosa consisting of vascular granulation tissue or of medullary fat with blood vessels (Fig. 2 and 24).

Third. — Thin spicuoles of bone frequently not calcified and lying most often within the choroid or beneath the pigment epithelium (Figs. 12 and 28).

GENERAL DESCRIPTION OF BONE

The compact portion of a bone nearly always showed strata of different density, whether or not calcification had taken place. The various strata represented the method of growth, which was by deposition of a material that was neither connective tissue nor hyalin, but a specific preosseous substance, in which as a rule there were no bone corpuscles. As a rarity there was a definitely regular lamellation, reminding one of the concentric lines seen in a cross-section of a tree which grows also by deposition of one layer on another (Figs. 30 and 33). Never was it supposed that the lamellation in any way represented a transformation of connective tissue into bone. The lamellae of bone matter seemed to be the product of a process of deposition. There was never anything resembling a condensed connective tissue capsule at the periphery of the bone, but it always lay naked and sharply defined.

BONE CORPUSCLES

The following two types of bone corpuscles were differentiated:

1. Primitive bone corpuscles (Fig. 37). These lay in very small lacunae that were rounder and more polygonal than those of normal bone. The cells showed but few processes, and on the whole appeared very simple. Haversian canals were correspondingly few, represented by short tubes. The corpuscles of this type had the markings of being in an embryonal stage.

2. Fully developed bone corpuscles (Fig. 30). These were of the typical star-like shape seen in normal bone. The canaliculi that accommodate cell processes revealed themselves as delicate thread-like coils. They were more numerous than the canaliculi of the primitive bone corpuscles, 10 to 20 originating from a single lacuna.

Bone corpuscles differed in various parts of the same bone formation, at some points taking on an embryonal type and at others being fully developed. The presence or absence of calcification had no effect on the type of corpuscle. It happened that in one instance in the same plate of bone both primitive and well developed corpuscles were found (Fig. 30). Strange to say, the latter were external to the first, which did not seem logical because in the

process of deposition the center of the bone should be the older and therefore contain more highly developed corpuscles.

BONE MARROW

Bone marrow was associated with the second type of extensive shell-like formation at the papilla (Figs. 2 and 11). It usually lay enclosed in an elaborate reticulum of long slender bone trabeculae lying parallel to the surface and connected with one another by still more delicate bridges of bone. The marrow was made up of a mesh work of granulation tissue, in which scattered lymphocytes or lymphocytic nodules were found, together with many endothelial tubes. In the interstices of the meshwork a large amount of fat cells was found forming typical yellow bone marrow (Figs. 24 and 33). Fat in the globe is associated with bone trabeculae and almost never occurs independently.

Marrow was never encountered in the flat and spicuole types of bone which remained one of the cranial bones. The presence and the amount of marrow was directly related to the amount of bone.

In one special case (Fig. 24) there were relatively few bone trabeculae but these enclosed a large cavity about 5 mm. in breadth which was filled with fat marrow. It is remarkable that fat in such a large amount could have been found because the globe normally contains no fat. The presence of fat pointed the way to the conclusion that intraocular ossification represents not a degenerative process but a true metaplastic proliferation. To illustrate this, in a papilloma the primary proliferation is that of the epithelium, but accompanying this there is also a proliferation of connective tissue which provides nutrition for the multiplying epithelium. Again, a carcinoma is by no means solely a growth of the epithelium but there goes with it a proliferation of the connective tissue and both together form the tumor. In an analogous way one should consider the development of the bone marrow spoken of here as essential to the nourishment of the trabeculae of spongioid bone. Probably bone marrow fat is different from the usual type of fat found elsewhere in the body, the latter serving as a storehouse for general nutrition.

VASCULAR SUPPLY OF BONE

Bone was frequently found in tissue abundantly supplied with blood (Fig. 27). This was particularly true of the third type of

bone, in which platelets of bone were found embedded in the vascular choroid, or between the pigment epithelium and choroid, in the neighborhood of the papilla, where the best blood supply of the entire globe is to be found. The second or spongioid type often showed large blood vessels traversing the trabeculae or in the marrow. These vessels often resembled large endothelial tubes and cavernous spaces, sometimes filled not with blood but with coagulated serum (Fig. 24). Inasmuch as lymph vessels are unknown in the globe it seemed likely that these were blood vessels in which blood clots had retracted.

One case of metastatic ophthalmitis after mastitis was noteworthy. A number of endothelial tubes, entirely filled with coagulated fluid, were present in a great quantity of fat marrow and not a single red blood corpuscle could be seen in them (Fig. 24). Recalling again that in normal globes there is no fat, whereas here there was so much of it, it would not be astonishing if lymph spaces had actually formed in this unusual case. Blood vessels of the spongiosa as a rule sprang from those of the choroid.

LOCALIZATION OF BONE

Certain places showed a definite predilection for bone. Sometimes there was just one isolated piece of bone. In other cases the bone was distributed on more than one place and this was termed "combined localization." The most common localization was found to be on the surface of the choroid, in the posterior quadrant of the globe, as is mentioned in the literature. From this localization it more than once extended forward over the equator to unite with a ring of bone that grew backward from the ora serrata. In the coalescence of the two zones a complete cup of bone was formed (Fig. 11). A small hole posteriorly was invariably left for the passage of the atrophic nerve fibers. The ring of bone at the ora serrata, which was generally not so complete as the one surrounding the nerve head, often possessed irregular jagged margins from extensions inward along cyclitic membranes (Figs. 3 and 4). As is well known, the growth of bone may be so extensive as to occupy almost the entire cavity of a shrunken globe.

Bone on the surface of the choroid occasionally sent slender prolongations or lamellae into the underlying choroidal stroma (Fig. 15). More rarely bone within the choroid sent prolongations upward to cover the surface of the overlying pigment epithelium (Fig. 20).

OSSIFICATION OF THE LENS

Bone formation within the lens was encountered 7 times (Figs. 11 and 34) and a few times bone was found in the circumlental space. In every instance whenever bone was found in the lens, on the ciliary body, or at the ora, it was always combined with bone elsewhere.

Ossification of the lens is best explained in the mode accepted in the literature, that is, by the extension of connective tissue into the capsule. This may happen when no injury has destroyed the lens capsule. In a case of metastatic ophthalmitis in the third month of life, toxins dissolved the posterior capsule of the lens, permitting connective tissue to enter from a cyclitic membrane. That really connective tissue was the basis of the ossification and not epithelial fibers derived from the capsular epithelium was proven by the presence of blood vessels in the midst of bone lamellae that substituted lens matter. It was interesting that in several instances islands of pigment epithelium presented themselves among the bone trabeculae within the lens. These could be traced as coming from the ciliary epithelium which had proliferated and formed long strands extending inward from the flat part of the ciliary body.

The possibility that ossification takes place within the lens capsule more readily than in a cyclitic membrane was more than once suggested. One case was very striking in that bone was confined entirely within the capsule, and the membrane that connected the capsule to the ciliary body was free of bone.

BONE ON THE CILIARY BODY

Bone on the surface of the ciliary body as a rule did not give the impression of having been a transformed connective tissue membrane. The bone lamellae always lay on the surface of the epithelium itself and more than once even separated a membrane from the ciliary body. As a matter of fact direct evidence of the transformation of a dense connective membrane into bone was lacking everywhere as a general thing. On the contrary, a narrow stratum of loose fibroblasts often separated the bone from a denser connective tissue membrane (Fig. 16).

In a special case (Fig. 35) the lens matter had been destroyed and the almost empty sac, containing a few debris of lens fibers, was fixed to the iris by a membrane. At the same time the ciliary processes were drawn to the capsule, behind which a shell of

bone lay upon the cyclitic membrane which surrounded the remains of the lens.

OSSIFICATION OF THE RETINA

The retina showed bone formation in 5 cases. In 2 of these extensive ossification of the choroid was combined with small pieces of bone in the retina near the ora serrata. The ora is a favorite site for different kinds of reactive proliferation, as for instance, migration of the pigment epithelium into degenerated and detached retinas. It is therefore not astonishing that bone was found here at the retinal attachment to the ora serrata.

Bone within the retina differed from that upon the choroid, presenting more jagged outlines and being more irregularly calcified. The bone corpuscles were much scantier, rendering the differentiation from pieces of chalk difficult. In 2 other cases noncalcified bone was found lying on the retina. In each of these the retina was detached, and a circumscribed adhesion extended between it and the choroid (Fig. 18).

An unusual case was that of the globe of a 33 year old woman which became blind in early childhood. Microscopically there is seen at the place of adhesion between retina, sclera and ciliary processes a small, isolated piece of calcified bone with well developed bone corpuscles. As in the immediate neighborhood, a membrane developed, it is likely that traction acted as a stimulus to produce bone (Fig. 6). But the bone was not produced by the solid connective tissue membrane which is found some distance away, only delicate prolongations of it being near the bone.

SITES OF PREDILECTION FOR OSSIFICATION

A comparison of the distribution of the bone in various parts of the globe brought out that there are two favorite sites of predilection:

1. At the posterior quadrant, especially around the papilla, as shown in Figs. 1, 10, 22 and 23.

2. At the ora serrata, as shown in Figs. 3, 4, 6, 7 and 22.

Predilection for the region of the papilla held good as well for small pieces of bone in the choroid as for large shells of bone on its surface. It was at the papilla that large shells of bone attained their greatest thickness. Sometimes, pulled forward by traction, the detached retina dragged the papilla and the adjacent choroid with it, and in the drawn out atrophic part of the choroid, bone was sometimes found (Fig. 10).

Bone in the immediate neighborhood of the papilla lay not only on the surface of the choroid, but also frequently in the choroid itself, in uninterrupted contact with the optic nerve (Figs. 23 and 25). In one case the optic nerve and retina were pulled forward and in another case the reverse happened, the papilla being pushed backward into a glaucomatous excavation. In either instance the point was that traction was exerted on the adjacent choroid (Figs. 10 and 25).

At the second place of predilection, the ora serrata, bone was found only when the attachment of the retina was confined to the immediate neighborhood of the ora serrata, by which is meant that the retina remained in direct contact with the choroid at this point (Figs. 3, 4 and 5). This condition could not have been fulfilled had the detached retina, by taking with it the ciliary epithelium, extended forward along the ciliary body. In this latter event only a delicate film of epithelial cells would have connected the detached retina to the uveal tract and there would have been no traction.

Bone at the ora was found chiefly upon the internal surface of the choroid and it was as a rule covered with pigment epithelium. Sometimes it was calcified and as often it was not. The "oral bone," as it came to be called in this study, at times formed a complete ringlet at the ora serrata. Then, again bone was missing on one side. It often included spaces filled with large vessels and containing islands of proliferated pigment epithelium (Figs. 3 and 4).

The "oral bone" generally had a triangular outline in cross section, with the base anchored to the choroid. It was to the apex of the ossified triangle, pointing toward the median line, that the detached retina was fixed, leading from here backward and inward to the interior of the eye.

In old cases of glaucoma bone was usually found immediately adjoining the papilla, either in or on the choroid. The predilection for the juxtapapillary zone was striking. Most likely the recession of the cribriform fascia by causing traction on the choroid played an important role in this localization (Fig. 25).

"Oral bone" was never found without the presence of bone elsewhere in the globe. In 19 cases it was found to be thus combined. It is interesting that in only 2 of the 19 cases was there an abundant ossification anywhere, for it usually happened that when there was relatively small amount at any one point, bone was present at different points. The reason for this was sought in the

extensive detachment of the retina that characterized these 19 cases, producing traction on the choroid at the ora and at the optic nerve, at the same time exposing the entire choroidal surface to toxins in the subretinal fluid.

ROLE PLAYED BY TRACTION

Traction was believed to be a most important stimulus to the production of bone in various parts of the globe. Different reactions in the choroid are attributed to the traction exerted by a detached retina or a cyclitic membrane, for example, lymphocytic foci and tumor-like proliferations of the epithelial layers at the ora. It is but a step farther to attribute the formation of bone also to traction. In support of this theory bone was present at other sites, where a detached retina, by means of adhesions, exerted traction on the choroid. Here proliferation of the neuroglia and formation of bone was not uncommon (Fig. 18).

a. *At the Papilla*

The characteristic juxtapapillary ossification seen in this material in connection with a retinal detachment or a glaucomatous excavation was attributed to traction, the detached retina pulling the choroidal ring forward or the recessed cribriform fascia pulling it backward and compressing it over the scleral ring. To be sure, traction does not give rise to bone within the nerve itself because the lamina cribrosa seems not to be a type of tissue able to produce bone.

It is interesting how it comes about that a detached retina may exert traction on the tissues in the immediate neighborhood of the disc. At the start traction causes inflammatory reaction, the disc becoming swollen and the adjacent choroid infiltrated. In time synechiae form between the papilla, choroid and retina. In this way traction from a tautly drawn forward retina may be the cause of bone formation in the collar of the choroid that embraces the nerve head (Figs. 10 and 23).

b. *At the Ora Serrata*

The bone found at the ora serrata was almost beyond doubt excited by traction exerted on the choroid at this point (Figs. 3 and 4). Seemingly no bone is ever produced at the ora when the retina falls completely away from the choroid carrying with it the ciliary epithelium. In such a case, instead of bone there is apt to be formed the "Ringschwiele" (callous) of Leber (Fig. 14).

This is an epithelioid connective tissue covering the flat part of the ciliary body from the ora serrata forward to the place where the detached ciliary epithelium branches off from the pigmented epithelium of the ciliary body. It is believed that "Ringschwiele" is a metaplastic proliferation of the pigmented ciliary epithelium under the influence of toxins in the subretinal fluid. It was striking that in 81 cases there was only one globe that showed a "Ringschwiele," which is not at all rare in general pathologic examinations. The explanation was that those cases in which the retina detaches with the ciliary epithelium, there being no traction, do not lead to ossification, and therefore they were not included in the present material.

The subretinal fluid, although it is assigned as a cause for the production of callous, does not seem to play the same rôle in the production of bone at the ora. Here the bone is within the stroma of the uveal tract and not on its surface as in a "Ringschwiele." Furthermore, oral bone was always covered by pigment epithelium which showed far less hypertrophy than it does in "Ringschwiele."

Lymphocytic infiltration at the ora in this series was all the more remarkable because most of the globes had been atrophic for a long time and ossification was always present. The lymphocytes were either scattered or disposed in well formed nodules, once even in the shape of a typical node with a germinal center. In some globes the infiltration probably represented remains of an original spontaneous inflammation. In one case (Fig. 36) there was an abscess at the pupillary border of the iris, enclosing a foreign body, all of which was surrounded by granulation tissue. This globe showed lymphocytic foci at many points in the entire uvea. The history spoke of spontaneous blindness in this eye coming on 20 years before enucleation. Apparently the patient was unaware of injury, and in spite of the long period of time, the foreign body kept up constant irritation. Ossification occurred in this instance at the typical places of predilection, namely, at the ora and at the papilla, and in addition on the surface of the ciliary body.

There were instances (Cases 13 and 18) in which lymphocytic infiltration was the result of recent irritation. However, a special place of predilection for lymphocytes was at the ora. The matter of localization of lymphocytes at the ora led to the conclusion that it was not the process of bone formation and the presence of bone lamellae that produced lymphocytic infiltration, but that both the

bone and lymphocytes were the effect of the same stimulus, namely, traction caused by a shrinking membrane or a detached retina.

Wherever an extensive spongioid shell of bone lined the choroid the pigment epithelium was correspondingly missing or fragmentary (Fig. 15). However, where platelets of bone were found in the choroid the epithelium was always present, at least to a certain extent (Fig. 28). The preservation of the pigment epithelium and of the choriocapillaries was considered proof that the underlying bone was not due to any action on the part of the pigment epithelium or of any membrane on the surface of the choroid. The pigment epithelium sometimes underwent changes, the cells becoming larger and flatter and the pigment granules less numerous, just as in many cases of ordinary chorioretinitis (Figs. 2 and 5).

THE LAMINA VITREA

The lamina vitrea was often recognizable, either on the external or the internal surface of bone. There were two instances in which the lamina lay within the fabric of a plate of bone, being covered by bone on both sides. Occasionally the lamina vitrea underwent a peculiar change as it ran between the bone and the choroid. In one globe (Fig. 5) it gradually attained great thickness becoming faint and fibrillated and finally disappearing in the tissues. In another case (Fig. 37) there was an enormous skein of coiled and folded threads of lamina vitrea apparently produced by splitting of the membrane and retraction of the fine threads.

AMOUNT OF OSSIFICATION

The amount of bone encountered was variable. Large spongioid formations were noted in 34 cases. In 44 cases bone appeared in isolated plates.

CALCIFICATION

In considering calcification in ossified globes one should bear in mind the possible distributions of chalk according to the kind of tissues:

First. — Calcification of bone itself.

Second. — Calcification of pre-existing tissues.

In 28 globes there was an absence of calcification anywhere. In 21 decalcified globes considerable amount of chalk had certainly been present, but it was not possible to give details as to its distribution. In spite of chalk present in bone or elsewhere it had been possible to make selections of 31 globes, in 5 of which calci-

fication was scanty and in 5 it was particularly marked. Calcification first made its appearance in small areas of bone (Figs. 8 and 12). No support was brought forth for the theory that chalk deposits serve as an impetus to the formation of bone. On the contrary, this study brought out that, first, bone is laid down and that, second, only later it becomes impregnated with chalk. Corroborating this theory, as stated above, the findings in 28 of the 81 cases studied revealed ossification without calcification.

It happened in the material that in addition to its deposition in bone, pure deposits of chalk occurred in various places, the most common being the lens. This was impressive when it was recalled that in 32 cases the lens was not available for study. In the decalcified group there was no chalk in the lens of 4 cases. In the cases of non-calcified bone calcification of the lens took place 5 times.

Chalk deposits were seen in only 5 instances and then not to any great extent. This is another proof that calcified membranes are not essential in the formation of bone. Whereas lime in a corneal scar often leads to expulsion by a sequestered inflammation not once was such a reaction visible in the intraocular membranes that contained chalk.

In 8 globes calcified drusen of the lamina vitrea were present in abundance. In 3 of these the lens was calcified.

The retina is known to be inclined to deposits of chalk. The reason may be that the walls of the blood vessels are prone to sclerosis. In this series chalk was abundantly deposited in the walls of the medium sized vessels and capillaries of a number of globes (Fig. 9). So great may be the deposit that the lumen may be occluded and appear as a small globule of lime in an atrophic retina. Altogether there were 9 cases of calcified retinas. In 3 calcification of the lens was noted and in 6 the lens was missing. Seemingly there was an element of parallelism between calcification of the retina and that of the lens.

Regarding the distribution of chalk within the lens, it was found more often in the anterior and equatorial lamellar layers than in the posterior parts. The chief metabolism of the lens is thought to take place in the area of the subcapsular epithelium. When in disease the metabolism is interfered with, it follows that here regressive metamorphosis first takes place. One extraordinary case showed calcification of the entire lens nucleus, after spontaneous iridocyclitis 10 years before enucleation at the age of 4 years.

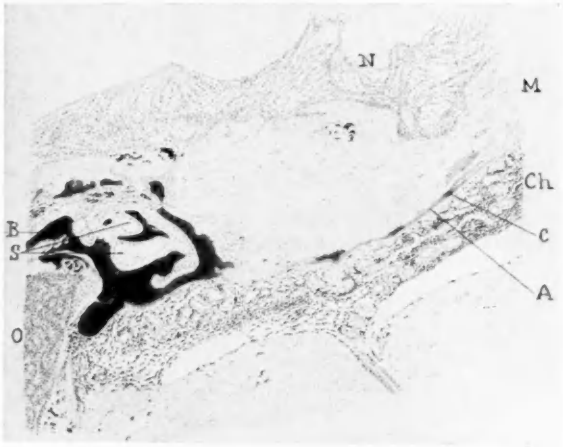

FIG. 1 — (Samuels) — Atrophic globe after cysticercus. Beginning ossification. The atrophic optic nerve (O) is surrounded and covered by calcified bone (B), containing empty vascular spaces (S). Adjoining the bone a large membrane (M) lies in direct contact with the choroid (Ch), the pigment epithelium having disappeared. Along the outer surface of the membrane there is a narrow homogeneous area (A) in which there are several foci of chalk (C). The loose neuroglia tissue (N) is what is left of the retina.

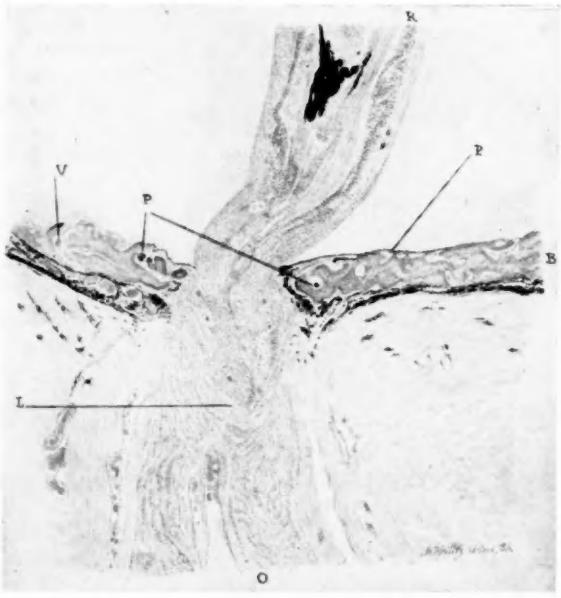

FIG. 2 — (Samuels) — Juxtapapillary ossification. The atrophic retina (R) is folded and contains masses of pigment. Lining the choroid is a layer of calcified spongioid bone (B) covered by pigment epithelium (P). Within the bone are cavities and canals containing blood vessels and proliferations of the pigment epithelium (P). The optic nerve (O) is very atrophic. The lamina cribrosa (L) is recessed, the large space in front being filled with degenerated retina and proliferated glial cells.

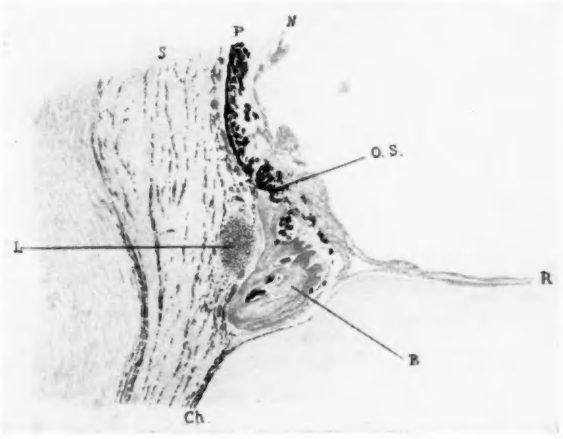

FIG. 3 — (Samuels) — Anterior to the ora serrata (OS) is a proliferation of the pigmented (P) and the non-pigmented epithelium (N) of the ciliary body. The detached retina (R) makes traction on a mass of bone (B) containing blood vessels and collections of pigment. In the choroid external to the bone is a large focus of lymphocytes (L). The lamellae of the suprachoroidal space (S) are separated by an albuminous fluid. The detachment of the ciliary body and choroid is due to traction exerted by the retina.

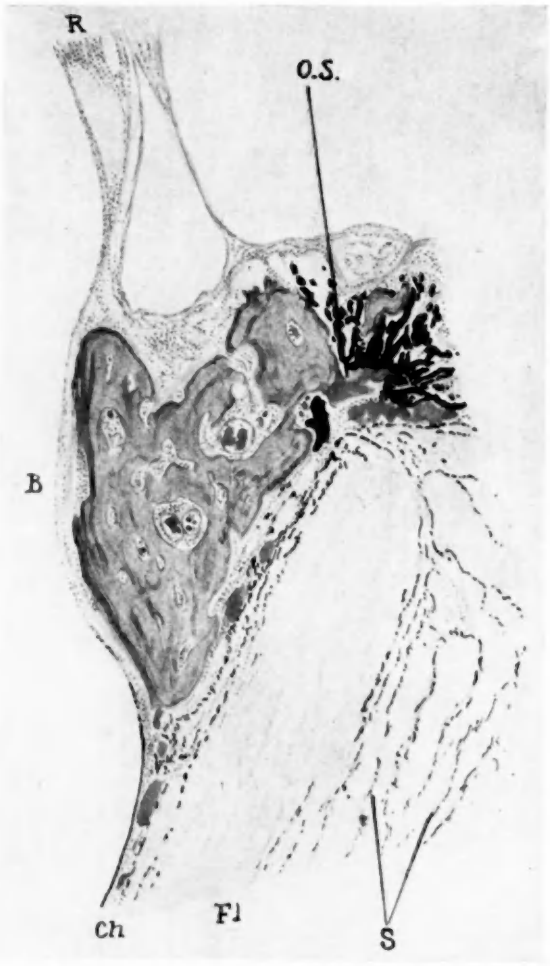

FIG. 4 — (Samuels) — Anterior to the ora serrata (OS) to the right the pigment epithelium is greatly proliferated. The retina (R) is put on the stretch forward and degenerated so that only a few strands remain. Posterior to the ora, a mass of moderately calcified spongioid bone lies on the choroid (Ch). In the substantia spongiosa are large blood vessels. Apparently the detached retina exerted traction on the bone. The choroid is detached by an albuminous fluid and the suprachoroidal fibers (S) are expanded.

FIG. 5 — (Samuels) — Degeneration of the lamina vitrae. A man 27 years. Incised wound at the age of 2 years. The drawing is from near the equator. The stroma of the choroid (Ch) is dense and is partly covered by a calcified shell of bone (B). The lamina vitrae (L) is undulating and becomes thick and fibrillated to the left. The bone is of spongioid character, a loose connective tissue occupying the cavities. The lamina vitrea is covered by the flattened pigment epithelium (P) which contains no pigment.

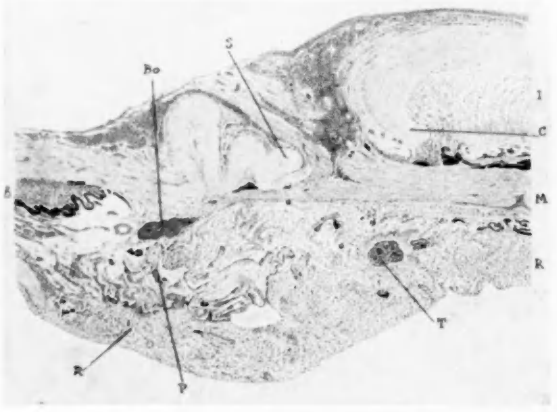

FIG. 6 — (Samuels) — Isolated fragment of bone next to scleral rupture. The corneal flap (C) is rolled inward, and the scleral flap (S) is curled on itself. Solid membranes (M) separate the iris tissue (I) from the convoluted retina (R) and close the wound. The ciliary body (B) is torn away from the scleral spur lying much farther posteriorly than it should. Anterior to the ciliary body is an isolated fragment of bone (Bo) on the surface of the sclera. Between the bone and the retina are remnants of necrotic ciliary processes (P). In the retina are darkly stained pigmented tubers (T) (proliferated epithelium).

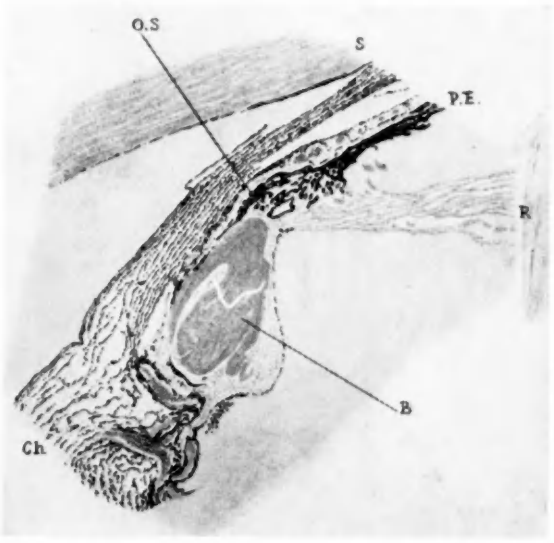

FIG. 7 — (Samuels) — Splinter of copper in eye for 15 years. At the ora serrata (OS) is an abundant proliferation of the pigment epithelium (PE) of the ciliary body. The highly degenerated retina (R), composed solely of neuroglia fibers, extends from the ora serrata (OS) toward the center of the globe. A large mass of non-calified bone (B) lies on the choroid (Ch) posterior to the ora. The choroid is detached and the lamellae of the suprachoroidal space are expanded. S — sclera.

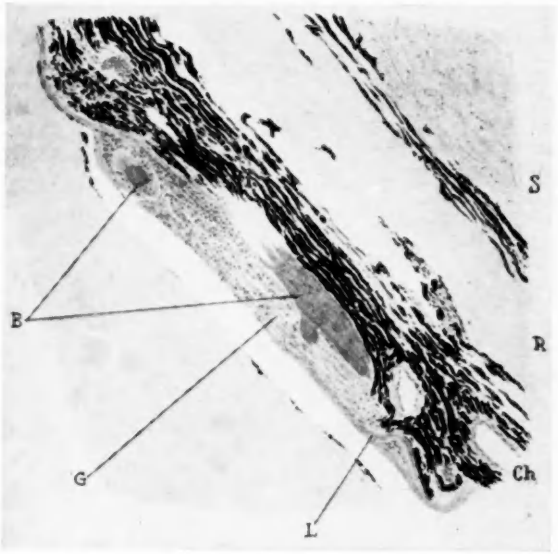

FIG. 8 — (Samuels) — Beginning bone formation between choroid and retina. Copper splinter in globe 15 years. The darkly pigmented choroid (Ch) is separated from the sclera (S) by albuminous fluid (A). There lies on the choroid, apparently beneath the lamina vitrea which is thickened at L, two small pieces of bone (B) which are at an early stage of ossification judging from the slight amount of chalk present. The tissue in which the bone is found resembles glial tissue (G). Only traces of the pigment epithelium are present.

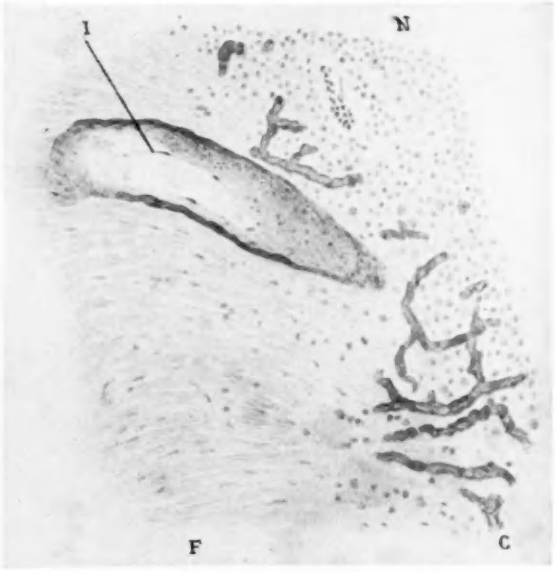

FIG. 9 — (Samuels) — Man 57 years. Spontaneous blindess. Atrophic and ossified globe. In the detached and highly degenerated retina the blood vessels are partially calcified. A large calcified vessel is shown, the entire wall being permeated with fine chalk granules, only the intima (I) remaining normal. To the left is the degenerated nerve fiber layer (F) and to the right the fused nuclear layers (N). Between the two layers is a field of calcified capillaries (C).

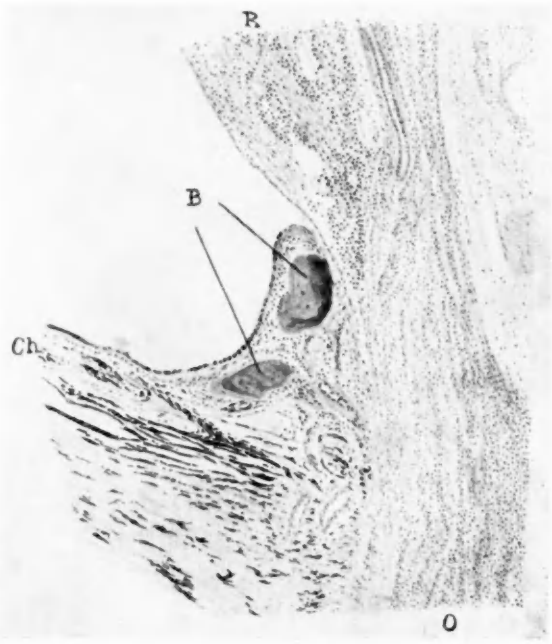

FIG. 10 — (Samuels) — Bone in the choroid near the papilla. The choroid (Ch) is adherent to the papilla and together with the degenerated retina is drawn inward. In the triangular space so formed are two inlays of calcified bone (B). The optic nerve (O) is atrophic. There is no sign of a membrane.

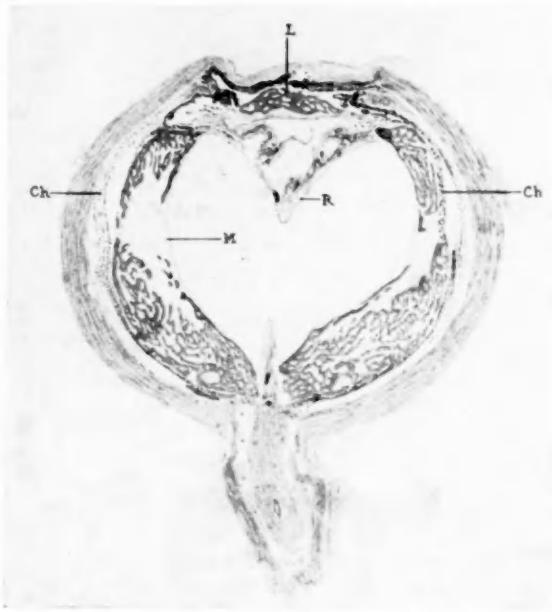

FIG. 11 — (Samuels) — Bone cup and ossified lens. Atrophic globe after a perforating eczematous ulcer. The entire choroid (Ch) is lined by a shell of spongioid bone, which is lined by a membrane (M). The retina (R) is highly degenerated. The lens (L) is replaced by bone which is separated by a membrane from the retina. Membranes on the ciliary body are present in other sections.

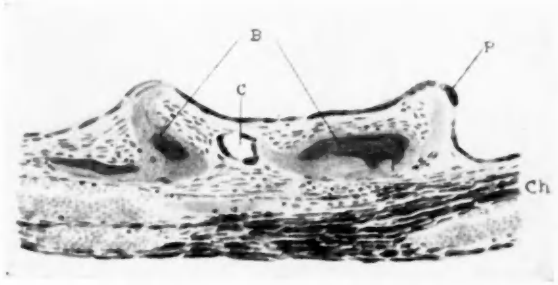

FIG. 12 — (Samuels) — Bone excrescences on the choroid. Injury 13 years ago. Embedded in connective tissue between the choroid (Ch) and the pigment epithelium (P) are 2 pieces of calcified bone. Lying between the two is a cavity (C) lined by pigment epithelium. The pieces of bone form a projecting mass over the surface of the choroid. The pigment epithelium (P) is made up of flattened cells and is poor in pigment. The choroid (Ch) is but little changed.

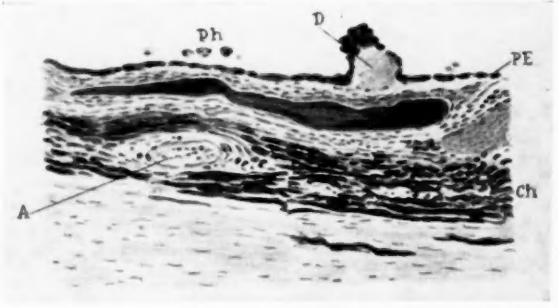

FIG. 13 — (Samuels) — Bone in choroid together with drusen on the surface. A sliver of bone, coated by a layer of fibroblasts, lies in the choroid (Ch) beneath the pigment epithelium (PE). A druse (D) projects over the bone, covered by proliferated pigment epithelium. A few phagocytes (Ph) filled with pigment lie on the choroid. At A a choroidal artery has almost lost its lumen.

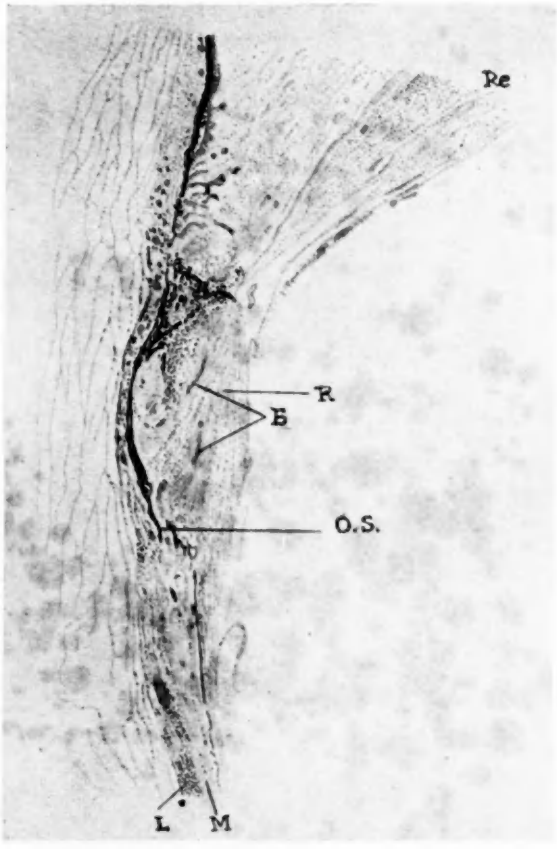

FIG. 14 — (Samuels) — "Ringschwiele." The ora serrata (OS) is recognized by the proliferation of the pigment epithelium. At R is a ring callous which resembles solid connective tissue and contains strands of non-pigmented epithelium (E). The degenerated retina (Re) is pulled forward. In the choroid is a membrane (M), to the left side of which is a focus of lymphocytes. At the opposite side (not shown) bone is present at the ora.

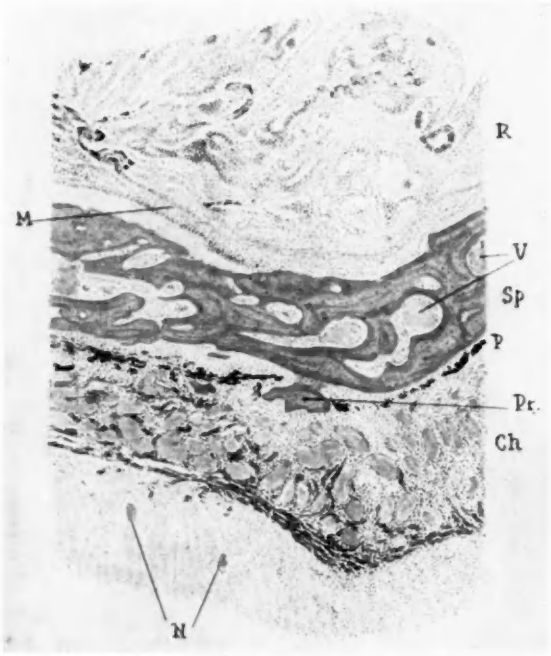

FIG. 15 — (Samuels) — Bone invading choroid. A phthisic globe 17 years after blenorrhoea neonatorum. The choroid (Ch) is much broader than nomal due to shrinkage of the sclera. Between choroid and degenerated retina (R) thick spongioid bone (Sp) is interposed, showing large vessels but no marrow. The bone lies in a bed of loose connective tissue. At one point it dips down by means of a process (Pr) through scattered pigment epithelium (P) into the choroid. In the sclera are atrophic ciliary nerves (N).

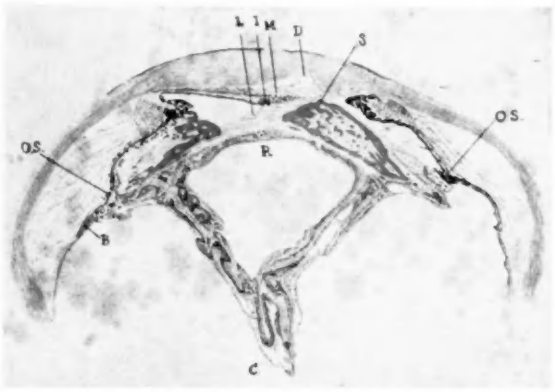

FIG. 16 — (Samuels) — Woman 59 years. Uveitis 15 years. Lens extracted 4 years ago. A membrane (M) fills the anterior chamber and leads to a scar. Of the lens (L) only fragments of the capsule remain. An infiltration of lymphocytes is in the pupillary area of the iris (I). On both sides a shell of spongioid bone with bone marrow (S) lies on the ciliary body. There is very little evidence of a membrane. Traction by the detached retina (R) is shown by its being fixed anteriorly and also by the wide detachment of the ciliary body and ora serrata (Os). A spicuole of bone (B) is seen in the choroid immediately behind the ora. Cystic spaces in the retina (C).

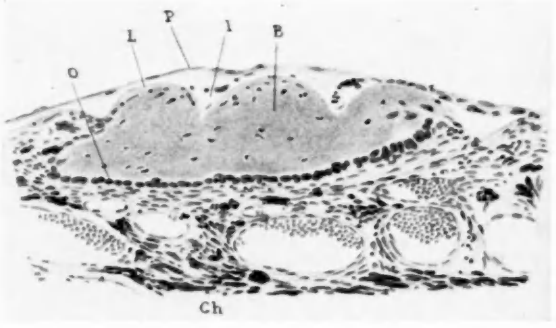

FIG. 17 — (Samuels) — On the surface of a practically normal choroid (Ch) is a piece of non-calcified bone, covered by the lamina vitrea (L) which conforms to the irregularities of the surface. A layer of pigment epithelium (P), containing no pigment granules, passes over the indentations (I) enclosing an albuminous fluid, containing pigmented cells and lymphocytes. Along the external surface of the bone a regular row of osteoblasts separates it from the choroid.

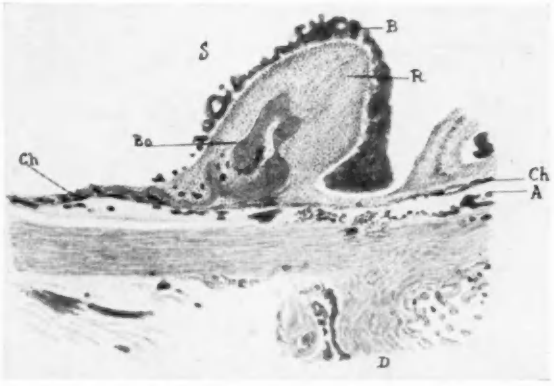

FIG. 18.—(Samuels).—Man 48 years. Glaucomatous eye, blind 18 years. A synechia (S) cut obliquely, extends inward from choroid (Ch). It is surrounded by blood (B). The retinal tissue (R) forms a fold and contains a non-calcified piece of bone (Bo). The choroid is somewhat detached by albuminous fluid (A) containing blood. Dural sheath of the optic nerve (D).

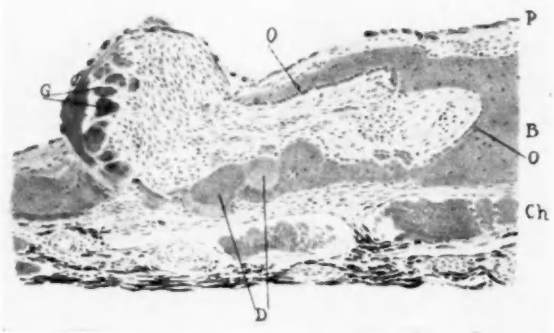

FIG. 19.—(Samuels).—Giant osteoblasts. A non-calcified piece of bone (B) lies on the choroid (Ch), covered by the pigment epithelium (P). At the periphery of the plate of bone (not shown) the lamina vitrea can be traced running between bone and choroid. The bone corpuscles seen are of primtive type. The large cavity within the bone is filled with fibroblasts. Rows of osteoblasts (O) line stretches of the cavity. In the projecting portion there are a number of multinuclear giant osteoblasts (G). The homogeneous masses are drusen (D), which demonstrate the former position of the pigment epithelium.

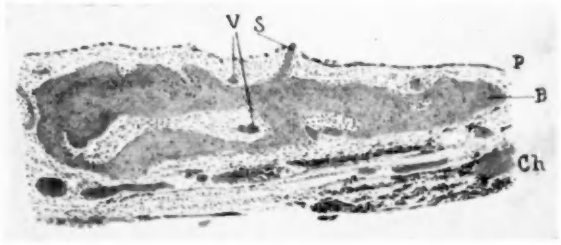

FIG. 20 — (Samuels) — Spicuole of epichoroidal bone. A non-calcified piece of bone (B) is embedded between the choroid (Ch) and the pigment epithelium (P). The lamina vitrea is not visible. The bone is of irregular configuration and contains lacunae filled with fibroblasts. It sends inward a sharp spicuole of bone (S) which elevates the pigment epithelium. Large blood vessels (V) within and without the bone speak for excellent blood supply.

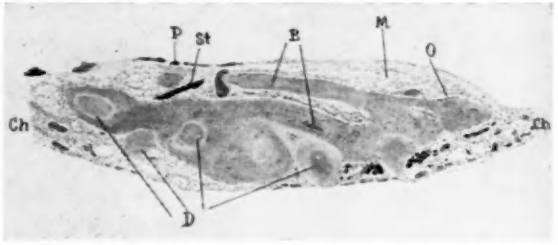

FIG. 21 — (Samuels) — Drusen embedded in bone. On the choroid (Ch) a large mass of non-calcified bone (B) is visible covered by pigment epithelium (P). Drusen are either embedded in the bone or are covered by it. At the greatest thickness of the bone the choroid has disappeared. The row of drusen marks the former position of the pigment epithelium. Osteoblasts are at O. A membrane (M) covers the bone. A strand of pigment epithelium (St) is in the midst of connective tissue and bone trabeculae.

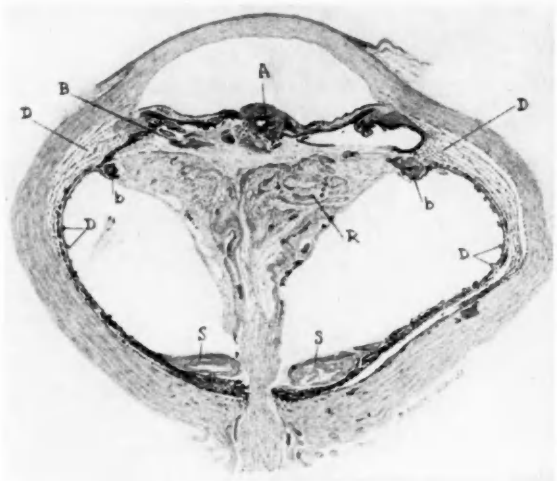

FIG. 22 — (Samuels) — Sites of predilection for ossification. An abscess (A) in the pupillary area represents the bed of a foreign body. Posteriorly the abscess is bounded by membranes and bone (B). The sites of predilection are shown in the drawing, namely, at the ora serrata and at the papilla. The oral bone (b) is between the ora and the retina (R). A shell of spongioid bone (S) surrounds the papilla. A number of drusen are scattered over the equatorial region. Traction of the shrinking retina is revealed by the detachment of the choroid. Drusen (D).

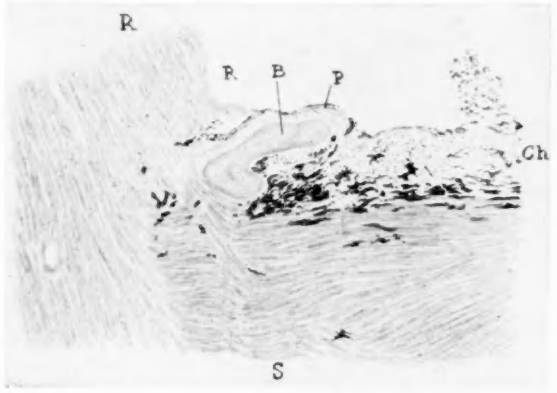

FIG. 23 — (Samuels) — The retina (R) and the optic nerve fibers (O) are drawn forward. A piece of decalcified bone (B), covered by pigmented epithelium, poor in pigment (P), is located at the papillary margin of the choroid (Ch). The choroid shows neither membranes nor inflammatory reaction. Sclera at S.

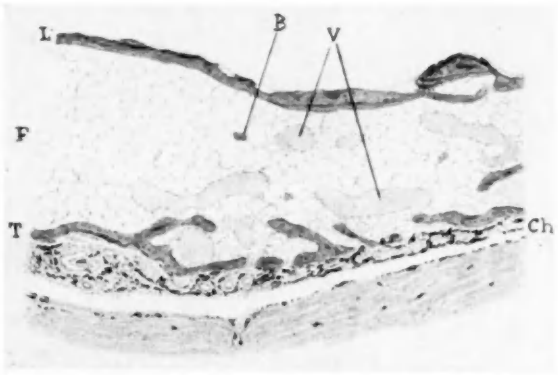

FIG. 24 — (Samuels) — On the thin choroid (Ch) is a spongioid type of bone with narrow trabeculae (T). The latter enclose a large mass of fatty bone marrow (F), in which are many vessels (V) containing serum. They consist of only an endothelial wall and are possible lymph vessels.

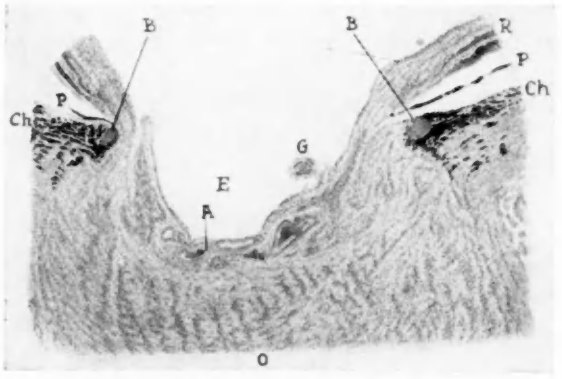

FIG. 25 — (Samuels) — A girl 17 years. Buphthalmos after injury at the age of 4. The papilla shows a deep wide glaucomatous excavation (E) on which a tuft of neuroglia (G) lies. Highly degenerated retina (R). On either side of the choroidal canal, embedded in the choroid, there is bone (B), circumpapillary ossification. No inflammatory reaction in the choroid is visible. The optic nerve (O) is completely atrophic.

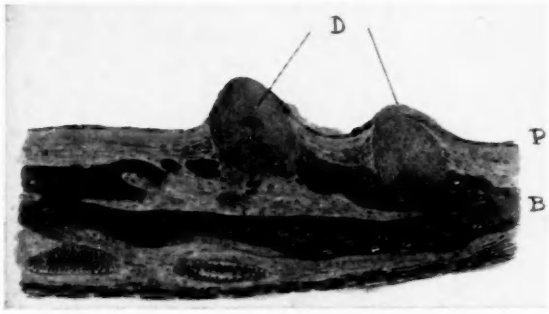

FIG. 26. — (Samuels) — Drusen and bone in choroid. Man 25 years. Injury 18 years ago. A thin shell of bone (B) lies in the choroid, next to the papilla. The pigment epithelium (P) covers the drusen which are unusually situated because they dip down into the stroma of the choroid instead of projecting over it. The pigment epithelium covering the drusen to the right has lost its pigment. The bone itself is embedded in very loose connective tissue fibers.

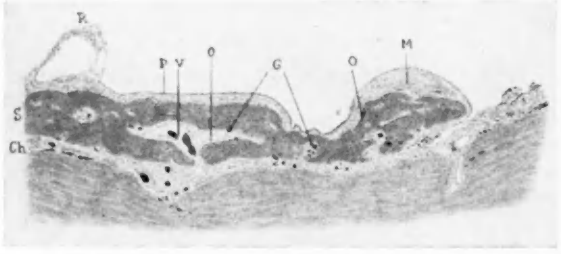

FIG. 27. — (Samuels) — The choroid (Ch) is covered by spongioid bone (S) which shows osteoblasts (O) at many places and also giant cells (G). The bone is covered by a membrane (M) which in turn is covered by pigment epithelium (P). The bone has developed at the side of a synechia (S) between choroid (Ch) and retina (R). The bone is well supplied with large blood vessels (V) extending from the choroid into it.

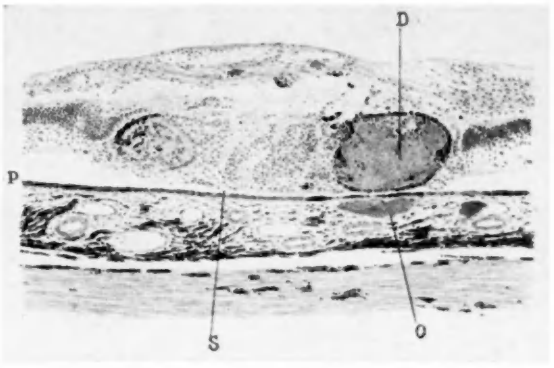

FIG. 28 — (Samuels) — A boy 14 years. Eye blinded in childhood. Corneal staphyloma. As often happens in this type of eye, a focus of degeneration and proliferation of the neuroglia (gliosis) happened at the posterior pole. A large hyalin drusen (D) surrounded by pigment lies in the external layers of the retina. Exactly beneath it and separated from it by the lamina cribrosa lies a spicuole of non-calcified bone. It contains bone corpuscles and shows a few osteoblasts (O). The retina is adherent at some spots (S) to the pigmented epithelium (P) which has lost much of its pigment content.

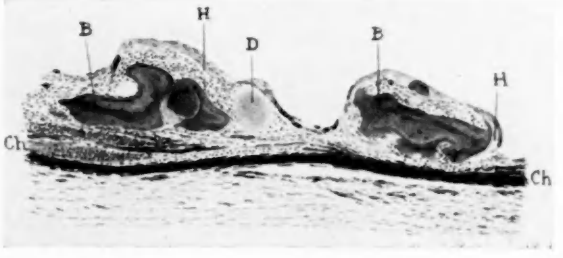

FIG. 29 — (Samuels) — A man 23 years. Eye blind after perforating ulcer many years ago. Overlying the choroid (Ch) are drusen (D) and excrescences of calcified bone (B). The latter contain an area of hyalin (H) and an area of calcification. Connective tissue and bone corpuscles are present.

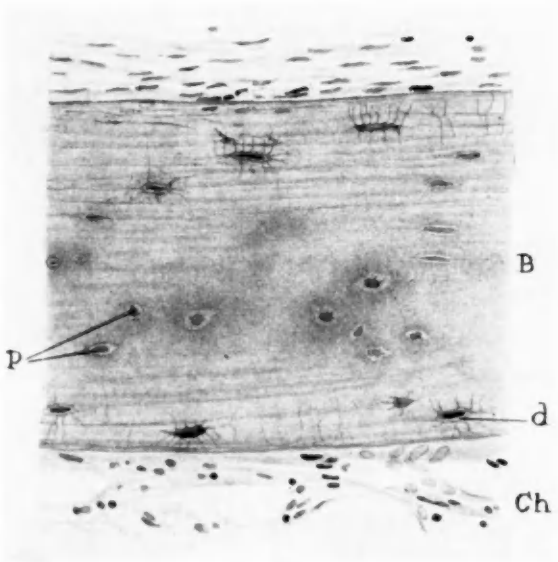

FIG. 30. — (Samuels) — Mature and immature bone corpuscles. A man 36 years. Injury 11 years ago. A piece of lamillated compact bone (B) lies on the choroid (Ch). Beneath either surface are well developed bone corpuscles (D) with long nuclei. In the center the corpuscles are plump and polygonal with few canals. They are primitive (P) and resemble embryologic bone corpuscles. The well developed elongated bone corpuscles lie parallel with the bone lamellae.

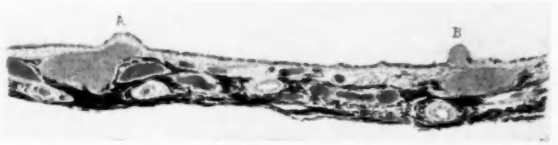

FIG. 31. — (Samuels) — Two spicuoles of bone lie in the inner layers of the choroid, one (A) bulges the pigment epithelium inward. The other (B) lies not far away. Above it, on the surface of the choroid, a druse is present. There are no membranes in the choroid and only a few fibroblasts surround the bone.

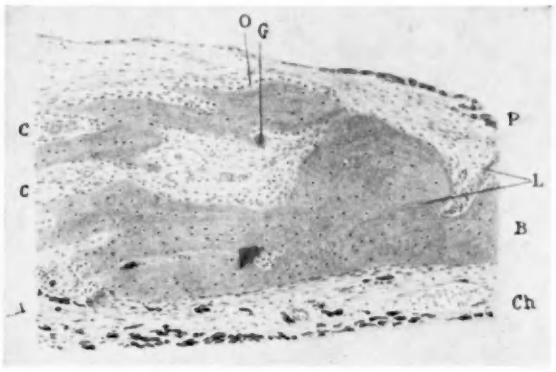

FIG. 32 — (Samuels) — Lamina vitrea between two bone lamellae. A spongioid non-calcified bone (B) is situated between pigment epithelium (P) and choroid (Ch). The lamina vitrea (L) is enclosed in the bone through which it may be traced. Loose vascular connective tissue (C) separates the bone lamellae. Only a few osteoblasts are seen and at one point a giant cell (G).

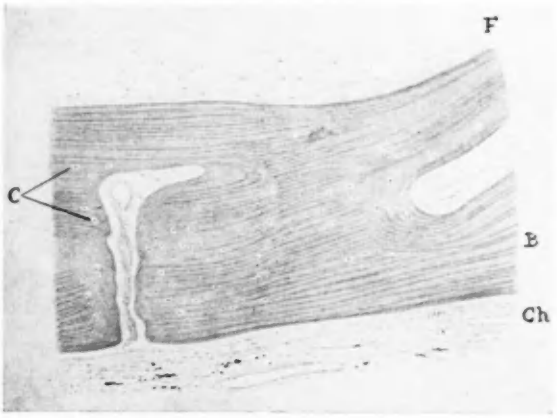

FIG. 33 — (Samuels) — Lamellation. The poorly pigmented choroid (Ch) is somewhat denser than normal. The spongioid bone upon it (B) shows marked lamellation. The bone corpuscles lie with the long axis parallel with the lamellation. The bone is covered by fat marrow (F).

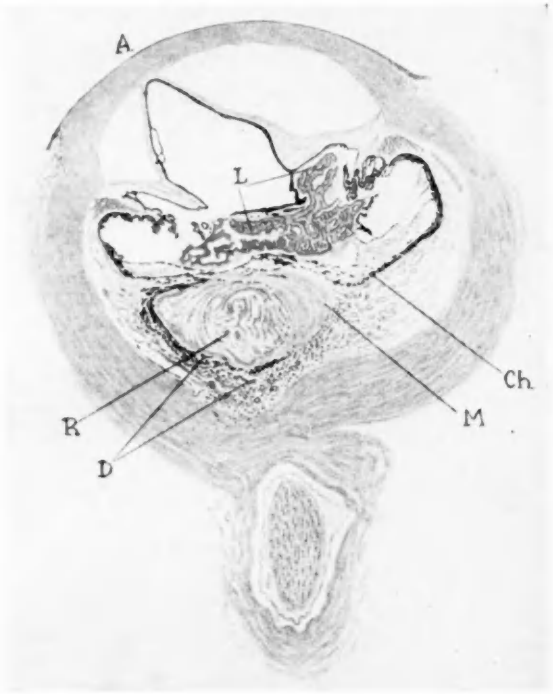

FIG. 34 — (Samuels) — Bone in the lens. In this phthisic globe the lens (L) is completely ossified. The ciliary body is free of membranes. The retina (R) is compressed and enveloped by a membrane (M) which has drawn the choroid (Ch) forward. In the choroid are several calcified drusen (D). At A an anterior synechia.

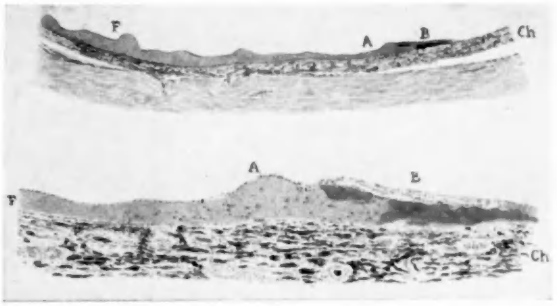

FIG. 35 — (Samuels) — Hyalin coating of choroid (sketch above). On the choroid (Ch) is a small piece of bone (B) which is calicified and which passes gradually into a homogeneous reddish area. The latter contains a few cells and finally gives way to a perfect fluid (F). The bone (B) is covered by a thin membrane; the fluid (F) by a layer of cells derived from the pigment epithelium.

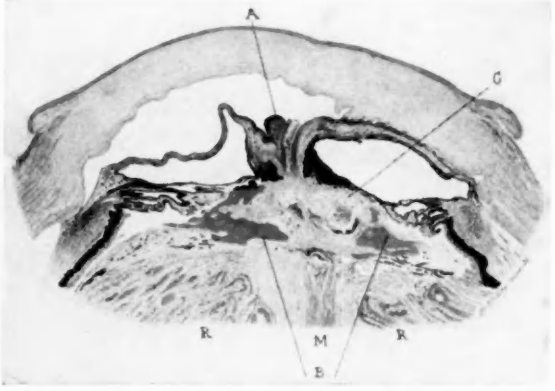

FIG. 36 — (Samuels) — Sketch of bone surrounding wrinkled lens capsule (C). A boy 19 years. Two years ago injury with a needle. In the center of an iris bombé is a small abscess (A). Of the lens only the capsule is left, to which the ciliary processes are attached. Iris, lens, ciliary processes, and retina (R) are all drawn together. The non-calcified spongioid bone (B) which surrounds the capsule shows no relation to any membrane on the ciliary body, at least on the left side. From the lens capsule (C) a membrane extends backward.

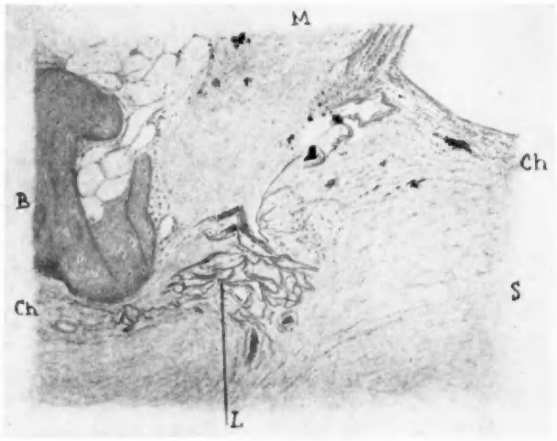

FIG. 37 — (Samuels) — Multiplication of lamina vitrea. The greatly changed choroid (Ch) lies on the sclera (S). Lying on the choroid is a large piece of spongioid bone showing bone corpuscles. A heavy membrane (M) extends forward from the choroid. The lamina vitrea (L) has disappeared in places and elsewhere is thrown into many folds.

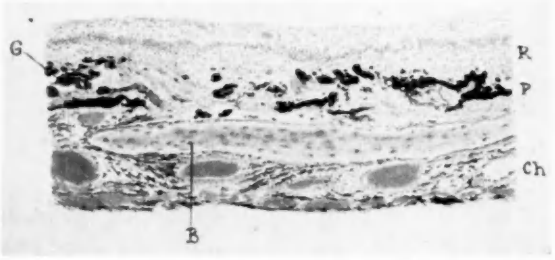

FIG. 38 — (Samuels) — Bone in detached choroid with well developed bone corpuscles. The choroid (Ch) and retina (R) are united by a large synechia. Among proliferated cells of the pigment epithelium (P) lie chalk concretions (C). Between choroid and retina is a plate of bone containing highly developed bone corpuscles (B). The choroid was to a great extent detached in this case.

ROLE PLAYED BY THE PIGMENT EPITHELIUM

In reviewing the list it was striking that practically never was bone associated with a membrane that did not contain within itself strands or islands of pigment epithelium. It seemed that those sites where the pigment epithelium has a prepensity to abundantly proliferate were especially inclined to bone formation. Accordingly bone was found in the immediate neighborhood of the optic foramen where the well-known choroidal ring of retinal pigment is situated. The region of the ora serrata is especially characterized by proliferation of the pigment epithelium, due, as is believed, partly to traction and partly to an inherent readiness of the pigment epithelium in this location to grow.

Concerning bone formation and drusen, this survey confirmed the findings of others that bone is mainly found at the posterior quadrant and drusen at the equator. At times bone and drusen were found side by side on the surface of the choroid. Bone was occasionally found enclosing drusen. The latter had no doubt been the first to form and only later was the bone that enveloped them laid down.

RELATION OF CONNECTIVE TISSUE MEMBRANES TO OSSIFICATION

In the literature emphasis is placed on the transformation of "membranes on the choroid" into bone. Treacher Collins[1] writes that "a new formation of fibrous tissue seems always to precede the appearance of the bone." Even when one admits that the mother cells of bone are of mesodermal origin, it is hard to get away from the fact that bone formation within a mass of dense connective tissue fibers would certainly be most extraordinary. It was frequently demonstrated in this material that bone may be found in the choroid free of connective tissue membranes or even of fibroblasts. Furthermore, although a membrane often lay on the choroid, yet ossification was far from being present in every membrane. Several times bone was separated from a solid membrane by a layer of loose areolar tissue.

It would be hard to imagine that spongioid marrow could develop by metaplasia from a solid membrane. It is easier to explain that spongioid tissue as a unit is a product of the choroidal elements. In corroboration of this statement it was noted how frequently bone lay directly on the choroid without any membrane intervening.

In the choroidal stroma membranes are rarely ever seen, but, notwithstanding, this bone was found in the choroid in 26 cases.

DEVELOPMENT OF BONE

Quoting from Parsons' *Pathology*,[2] it was Pagenstecher who in 1860 "proved that the bone originates in new-formed fibrous tissue or in normal tissues after they have undergone atrophic changes and that it grows in the same manner as the normal periosteal bone." To refute this age-old idea of the membranous derivation of pathologic bone was indeed the chief purpose of the present paper. Doubts as to the correctness of this early theory were aroused by the fact that one finds so frequently ossification without calcification.

Among the theories set forth as to the origin and development of intraocular bone are the following:

First. — The old conception that ossification takes place only in calcified membranes. In this material this type of development was far from being the rule. In one case (Fig. 1) there was a large membrane between choroid and retina, and here it did appear, as explained in textbooks, as if a focus of calcification at the edge of a membrane was the starting point of ossification. However, when one considered the number of large spongioid shells surrounding the nerve head, it was not possible to explain their development as having begun by ossification of a solid membrane. The question arose as to how can it be possible that a solid membrane becomes a substantia spongiosa occupying cavities between trabeculae of bone? It is not in accordance with pathologic knowledge that solid connective tissue should be thus expanded and transformed.

Second. — The theory of the development of intraocular bone from loose connective tissue, which is the one promulgated in this thesis. In the light of pathologic evidence in the survey this theory seemed to satisfy every argument. It is best understood by a study of bone formation found beneath the covering of the pigment epithelium. The latter probably plays a certain rôle in the process, not that the pigmented cells themselves produce bone, but it is most likely that they act as a stimulus to the connective tissue in their neighborhood.

In some of the atrophic eyes there were knob-shaped excrescences of the pigment epithelium containing a kind of granulation tissue derived from the choroidal stroma. In a similar way loose connective tissue plaques were sometimes found between choroid and pigment epithelium, and here in the midst of young connective tissue, bone was sometimes found (Fig. 12). Occasionally there

was so little connective tissue that it was scarcely visible. Of special interest was one case in which the choroid and retina were adherent and pieces of bone were found in the neuroglia of the overlying retina (Fig. 8).

OSTEOBLASTS

Osteoblasts were present in relatively few cases. They were distributed at circumscribed spots and were frequently separated from calcified bone by a narrow homogeneous zone. They had a large nucleus and an abundant protoplasm. They were disposed like cobblestones one against the other which influenced their shape, polygonal and irregular outlines being by no means rare, as with crowded plasma cells. The nucleus was often eccentric, lying closest to the bone. Giant cells sometimes occurred in connection with osteoblasts, lying at a short distance from the actual bone.

It is clear that osteoblasts were not the sole factor in the creation of bone otherwise they would have been seen far more often. Apparently the greater number of bone formations were laid down by fibroblasts or some other connective tissue elements. If one believes that a fibroblast can develop into an endothelial cell, as on the posterior surface of the cornea, and that in another place as a connective tissue fiber it can lay down an intercellular substance, then it is understandable that under pathologic conditions a fibroblast may lay down still another intercellular substance, namely, bone.

Altogether, osteoblasts were present in 22 globes, only 1 of which was calcified. In 6 globes osteoblasts appeared as giant cells, resembling foreign body giant cells. Osteoblasts were never situated in the neighborhood of old pieces of bone and never were they on the external surface of bone. They were mostly found lining cavities of young bone. That osteoblasts were seen as giant cells served as further proof that bone formation is not an expression of degeneration but rather one of proliferation. One was inclined to consider that osteoblasts indicated areas of growth or enlargement of bone. Speaking against this was the fact that in many cases bone lamellae were surrounded by a narrow homogeneous zone free of osteoblasts. Such a narrow zone was believed to be preosseous connective tissue, by the addition of which the mass of bone was being augmented.

Further support of the theory that active proliferation is the basis of all intraocular ossification was found in a case of spongioid

bone which lay in loose connective tissue, entirely in the choroid, beneath an intact pigment epithelium (Fig. 20). A small spicuole of bone sprang from the mass and pointed inward, elevating the pigment epithelium. A simple degeneration of connective tissue membrane never could have caused a projection of a bone spicuole, which showed its activity by elevating the connective tissue and the pigment epithelium.

Third. — The theory of the formation of bone from hyalin. Sometimes the impression grew that bone was directly formed out of hyalin by the migration of fibroblasts into a pellucid glassy substance, the fibroblasts being transformed into connective tissue. This was well demonstrated in a case of metastatic ophthalmitis. For a length of some 6 mm. the pigment was separated from the lamina vitrea by a perfectly homogenous substance. It was similar to the substance composing the physiologic drusen of old age (Fig. 35). It could be seen that gradually nuclei made their appearance along one side of the clear substance which here acquired a fine striated quality and along this margin deposits of chalk appeared. In this way a gradual transformation of hyalin into non-calcified and calcified bone apparently was being produced. It was only where calcification was present that a small amount of connective tissue covered the bone.

Fourth. — The formation of bone in a way peculiar to itself. This type was exemplified by small plates of bone, situated within the choroid near the papilla, which resembled the cross section of a nerve. Neither membrane nor irritation was visible in these cases. It must be that there is in this particular area a special phylogenetic property of the choroid to form bone directly, in response to a stimulus. This explanation harks back to the eyes of certain fishes in which the sclera is represented by a bony shell.

QUESTION OF STIMULUS

Granting that the greatest number of bone formations in this material had their origin in active proliferation of tissue derived from the stroma of the choroid, the question arose as to what the stimulus was that produced such activity on the part of the cells. In explanation there come to mind the following theories:

First. — The Theory of Stimulus Derived from Traction. That contracting and pulling membranes cause various tissues to proliferate is common pathologic knowledge. Proliferation of the pig-

mented and non pigmented epithelium of the ciliary body in response to the traction of cyclitic membranes is well known (Fig. 3). Under the influence of traction by an organized hemorrhage, the pigmented epithelium near a wound in the choroid may proliferate and form an epithelioid connective tissue, which has the property of drawing the edges of the wound together, thus functioning as connective tissue, a process which Treacher Collins termed "desmoplasia," desmo meaning to join.

Bone formation at the ora serrata and at the papilla is certainly to be considered as an example of metaplasia occurring in conjunction with traction.

Second. — The Theory of Stimulus Derived from Subretinal Fluid. In spontaneous cyclitis, toxic substances in the posterior chamber frequently produce abundant proliferation of the epithelial layers that line this space. In a similar manner toxic substances may develop in subretinal fluid in the course of time. It would seem that in practically every case of retinal detachment small sprouts of pigment epithelium develop sooner or later. These may become so marked that many types of pathologic drusen arise, such as excrescences of pure hyalin, calcified hyalin, vascular connective tissue, or large formations containing bone. This latter type throws strong light on intraocular bone formation in general, showing that it really is derived from vascular tissue that springs from the choroid. Ordinarily in a case of drusen at the equator the stimulus would be more likely a toxin than traction.

INHERENT QUALITY OF THE CHOROID TO PRODUCE BONE

Added to a stimulus there must be a certain inherent quality in the tissues of the choroid to produce bone, which is more marked at the sites of predilection, at the ora and at the papilla. In other words, at the places of predilection bone develops not only because of a stimulus, but because, that which is probably equally essential, the tissues here possess the power and tendency to produce bone. In the anterior chamber, although it is often almost filled with membranes and under constant irritation from toxins, yet no bone formation is seen, probably because it is not in the power of the tissues of the iris to produce it. In this research it was convincing that uveal tissue directly or fibroblasts derived from it are the primary cells in the production of ossification of the choroid.

Third. — The Theory of the Stimulus of the Pigment Epithelium. Recalling that it is just at the places of predilection for

ossification, at the papilla and at the ora, that the pigment epithelium is best developed, one is inclined to assign to the pigment epithelium a certain influence on the formation of bone. Clumps of pigmented cells were practically always seen in the cavities of the spongioid type of bone and in ossified lenses, confirming this view. It is to be borne in mind that there were plaques of bone found in the choroid, that contained no pigment, the overlying pigment epithelium showing no signs of activity. There was never any certain indication that from the pigment epithelium the fibroblasts were produced that were supposed to lay down the bone.

RELATION OF OSSIFICATION TO VARIOUS INTRAOCULAR DISEASES

The long accepted theory that ossification is the consequence of degenerative changes in membranes probably had its origin in the fact that a majority of ossified globes are atrophic or very much shrunken and contain many membranes, whereas ossification in distended glaucomatous eyes has been widely neglected.

It was interesting that in the material so few outspokenly phthisic globes showed ossification. According to the old theory phthisic globes, because of extensive membranes and because of remaining longer unremoved than atrophic globes, which are often early excised for fear of sympathetic ophthalmia, should be more frequently ossified and therefore have been more numerous in the tabulations. The reason of the relatively small number of phthisic globes was perhaps largely due to the fact that in these eyes the choroid, on which it is here argued ossification largely depends, is severely damaged or destroyed. If it were membranes that form the bone one would have expected to find a very high ratio of ossification in phthisic globes. Phthisis bulbi in pathologic collections is rare in comparison with atrophia bulbi, perhaps 10 times as rare. Nevertheless, the number of 5 phthisic globes in this large material was strikingly small.

The atrophic globes studied presented to a great extent detachments of the retina with large subretinal spaces containing an albuminous fluid. There were 47 cases of detachment of the retina, in 12 of which red blood cells were suspended in the fluid; in 5, phagocytes; and in 4, cholesterin. In 34 cases no subretinal space was present. To this latter group belonged the cases of glaucoma and chorioretinitis, and also 10 cases in which heavy, solid membranes filled the space between retina and choroid.

Of the 25 cases of endogenous inflammation that led to ossification, 6 suffered from metastatic ophthalmitis; 14 from spontaneous iridocyclitis; 2 from cysticercus; and 3 from spontaneous detachment of the retina. The metastatic cases all showed considerable bone formation.

GLAUCOMA AND OSSIFICATION

Only a certain percentage of the glaucomatous globes had increase of tension at the time of enucleation. Frequently glaucoma had given place to atrophy from such causes as late infection or organized hemorrhage. This was the explanation of why in 5 glaucomatous cases bone with a marked spongiosa was present. Bone in actively glaucomatous eyes was to a great extent of the type that contained small plates of non-calcified bone (Fig. 28). Of the glaucomatous globes 11 showed plates of bone at some distance from the nerve head and 9 showed juxtapapillary ringlets of bone.

It was striking that of the glaucomatous cases 13 showed bone within the choroid itself. The fact that there were 26 cases in the entire collection in which bone lay in the choroid is additional and convincing proof that the choroid is especially prone to produce bone directly of itself when it is subjected to abnormal pressure.

In 8 of the glaucomatous cases old chorioretinitis was manifest by the presence of adhesions between the 2 layers, and by peculiar changes in the retina which Leber described as gliosis with symptomatic pigmentary degeneration. Gliosis with symptomatic pigmentary degeneration is often associated with secondary glaucoma when the retina has been subjected to pressure, traction, or inflammation, as in cases of staphyloma of the cornea, buphthalmos and traumatic cataract with glaucoma. Of these 8 cases, 6 had increased tension at the time of enucleation and 2 had undergone atrophy. In each case of this group the ossification consisted of platelets of bone.

HEMORRHAGES

There was no instance of a great quantity of blood pigment, probably because most of the globes had been diseased for a long period so that the pigment had been dissolved in membranes. In 12 cases blood was found in the subretinal fluid, but in every instance the hemorrhage was recent as compared with the time required for the formation of bone. In one case a mass of bone cov-

ered the choroid, except for a small stretch, over which a subretinal hemorrhage lay. The underlying choroid showed dense lymphocytic infiltration. In this case one imagined that the hemorrhage had hindered the formation of bone. The general conclusion was reached that neither hemorrhages nor cholesterin crystals are of importance in intraocular ossification.

DRUSEN

In 42 cases drusen of the lamina vitrea were present. The number of drusen varied greatly from one to many in a slide. The bulk of most of them was made up of hyalin. In 10 cases calcification had taken place. The drusen were sometimes very small; at other times large and plump. By no means were they always exposed to free subretinal fluid, being now and then covered by bone and at other times by connective tissue membranes.

Inasmuch as drusen were most frequently located in a zone anterior to the equator, and inasmuch as bone was found mainly at the posterior pole, it appeared that the anterior part of the choroidal tract may possess a constitutional quality of producing drusen.

Whenever drusen were found covered by bone or by membranes it was considered that the former represented a primary and the latter a secondary reaction (Figs. 20 and 22). An unusual appearance was present in 1 case (Fig. 26) in which drusen, instead of projecting over the internal surface of the choroid, dipped down into its stroma. Although the lamina vitrea was not present in these cases there was no question but that bone and drusen lay within the choroid. Here again was presented another convincing evidence that no preexisting organized exudate upon the choroid could have become ossified.

From another standpoint drusen were interesting because they were found not so rarely overlying bone that was in the choroid (Figs. 28 and 31). Once a typical proliferation of pigment epithelium encircled a druse and immediately beneath was a large plate of bone in the choroid. In a similar case (Fig. 28) a large druse in the retina lay immediately over a piece of bone in the choroid. Presumably the irritation that produced a druse in these 2 cases at the same time produced bone in the choroid.

In addition to drusen there were found on the choroid peculiar excrescences filled with connective tissue and also homogeneous masses in which it was impossible to say whether one was dealing with hyalin or with coagulated fluid rich in albumin.

SYMPATHETIC OPHTHALMIA

An endeavor was made to ascertain what pathologic evidence there is as to whether or not ossified globes are especially dangerous for sympathetic ophthalmia. In the material of 81 ossified globes the direct question of sympathetic ophthalmia came up 6 times, because of inflammation in the fellow eye.

There was not one of the 6 globes pathologically suggestive of sympathetic ophthalmia, and there was never strong clinical evidence of it. Nevertheless, clinically one is justified in referring to the inflammation in the fellow eye as sympathetic irritation, and under the circumstances one should urge the removal of the ossified globe in order to be sure of being on the safe side.

According to this study, it may be affirmed that the danger of sympathetic ophthalmia from globes ossified after perforation is practically non-existent. It is instructive to recall that in an examination of over 100 globes of sympathetic ophthalmia there was not a single instance of ossification.[3] It must be extremely rare, if it ever does occur, that ossification and sympathetic ophthalmia happen in the same globe. Surely not after sympathetic inflammation has set in can bone develop, practically all uveal tissue having been destroyed.

Turning to sarcoma of the choroid, the writer ventures to affirm that atrophic and shrunken ossified globes are as little likely to develop sarcoma as they are to develop sympathetic ophthalmia. This statement is based on the fact that in an examination of 84 globes with a necrotic sarcoma there was not an instance of ossification, it mattered not whether the globe had become atrophic before sarcoma developed or had become so afterwards.[4]

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DISCUSSION

Dr. Brittain Ford Payne, New York: It has been my good fortune to study most of the specimens presented by Dr. Samuels. From his masterful work on *Ossification of the Choroid* at least two observations may be made:

Bone formation in the choroid has little or no bearing on sympathetic inflammation of the other eye. This seems startling in that Fuchs and others advise immediate removal if any sign of irritation or tenderness is present.

The second observation is that bone formation is absent in cases of necrotic intra-ocular sarcomata. This led to an investigation of other intra-ocular neoplasms in the collection of the New York Eye and Ear Infirmary. It was observed that bone was absent in sarcomata and carcinomata, whether necrotic or not. Necrotic gliomata of the retina showed calcium deposits.

An examination of specimens of ocular tuberculosis and syphilis failed to show bone formation. Hyalinization and calcification was not uncommon.

The architecture and anatomical features have been beautifully described. The development and location of the lesions explained. But the actual cause of bone formation remains as much a mystery as it was a century ago.

Physical, chemical and biological factors must be involved. It is possible that stress and pull on membranes cause bone formation, but it must be remembered low carbon dioxide tension may be a factor. Saponification of fatty acids as shown by Klotz may offer some explanation. All of these agents with others more complex probably give the answer.

We know from repeated investigations that hyalinization, calcification, and bone formation occur in dead or devitalized tissues. MacCallum tells us that the kidney, lung, spleen, liver, heart, blood vessels and subcutaneous tissues may be affected. No definite explanation has been found for these changes in any of the organs named. Ophthalmic literature fails to give a definite clue.

Fortunately, such heteropiasia is self limited in the eye. If bone should extend through the choroid and sclera and involve the adnexa, the astonishment would be great and the patient uncomfortable.

The question finally arises, should blind atrophic eyes be removed? They should be enucleated if pain, irritation, or the other eye is affected.

Dr. Mary Knight Asbury, Cincinnati: In reviewing several hundred eyes examined microscopically within the last three years, I was surprised to find bone in more than 10% of them. The series was much smaller than the essayist's, but bore out his findings in detail — the predominance in males (nineteen males and four females in my series), the wide age

distribution (at the time of enucleation the youngest was three years old, the oldest sixty-five), the types of bone found, the location, etc., and in addition furnished interesting exceptions, of which I shall speak later.

A critical and exhaustive study of the subject, such as Dr. Samuels has made, was needed to clarify our conception of ossification in the eye. The abundant literature consists largely of uncorrelated case reports, and the text books make scant mention of the subject.

I agree with the author that neither calcium nor visible membranes are necessary preambles to the formation of bone in the eye. Bone has been found in scar tissue, in cyclitic membranes, in eyes which also contain calcium deposits, but it is also seen in the choroid beneath the pigment epithelium, with little evidence of scar tissue formation around it. However, all the eyes in which bone is found have been severely injured or inflamed, and at least a minimal fibroblastic response is present. When the versatility of mesodermal cells is remembered, the production of bone ceases to be so mysterious. The young cell, which usually produces fibrous tissue, may find the conditions favorable for bone formation and develop that potentiality.

But the real issue is what constitutes the stimulus to bone production. Dr. Samuels favors "traction."

Let us return to the exceptions afforded by my specimens. The author describes "oral bone," an osteoid deposit in the region of the ora serrata, and states that one was never found without the presence of bone elsewhere in the globe. A small "oral bone" was found in the eye of a child, age four years, who had had vitreous abscess complicating meningitis eight months before enucleation. (This is the shortest duration of a lesion in which bone was found in this series.) No evidence of ossification was found elsewhere in the eye. In another eye the only osteoid tissue found was in the anterior part of the globe in the midline in a mass of scar tissue between iris and detached retina. In this instance the tissue immediately surrounding the bone was loose and very vascular, as described by the essayist.

Both malignant melanoma and bone were found in one specimen. The eye had been blind for twelve years because of retinal detachment of unknown etiology. The eye was removed because it had recently become painful, but the presence of tumor was unsuspected. The neoplasm was flat and had not broken through the lamina vitrea. It certainly was of recent development, and could not have been the cause of the old detachment of the retina. Elsewhere the choroid showed the changes characteristic of old detachment — drusen, foci of lymphoid infiltration, and moderate amount of bone arranged in thin plates in the inner layers of the choroid in the posterior half of the globe. Ossification was a sequel of retinal detachment, but I do not believe it was the cause of neoplasm.

I also doubt that ossification has any real bearing on the cause of sympathetic ophthalmia, but my series was less conclusive on that point than the author's. An eye had been shrunken for fifteen or twenty years, no further history was obtained. The retina had long been detached, the lens was calcified, and there were course granular deposits of calcium

on the surface of the ciliary body. A plaque of bone was found in the choroid at the disc margin and in the adjacent retina. Much of the choroid had been destroyed, but there was active inflammatory infiltration of the remainder, which was suggestive of sympathetic uveitis. Dr. Terry saw these sections about a year ago and concurred in the diagnosis. Recently a small shrunken globe was examined which had been blind for forty years. A thick shell of spongioid bone filled much of the vitreous cavity. Contrary to rule, the bone was thicker anteriorly and the shell was incomplete around the disc. The calcareous lens was embedded in fibrous scar tissue. In the center of the dense band occupying the anterior chamber there was a large plaque of osteoid tissue. The essayist states that ossification is never seen in the anterior chamber. The remains of the uveal tract was heavily infiltrated with inflammatory exudate resembling that found in sympathetic uveitis.

I am in agreement with many of the author's conclusions, but thought it might be of interest to note these exceptions to his findings.

Dr. C. B. Walker, Los Angeles: After conference with Dr. Samuels on this particular point, I am convinced these types are not prone to sympathetic ophthalmia. I venture to show the way they can be handled with prosthesis on the inside of the sclera and the conjunctiva.

This case having been made available by Dr. Boyce at the County Hospital where he is chief, illustrates the mechanism of inserting a glass ball in the place of the contents of the eye and it can be done without cutting a muscle or muscle sheath or any of the vascular supply, and therefore without disturbing the motion of the eye.

Several have tried to put various forms of prosthesis within the eye. Dr. Wiley of Los Angeles has used it with the bone sphere inside the eye, inside the sphere, but doesn't seem to have much success with it.

The thing that I have done to make the picture a little better is to remove the cornea. The cornea will stay transparent in case it is in good shape, and we hoped it might stay that way.

The ease with which these eyes will turn over, permitting desiccation from behind, is mainly illustrated in this picture. They turn very easy on their axis. It is turned here more than once so that you get an idea how easy it is to turn the eye and present it for interior work, without cutting muscles.

That represents an early picture where muscles were cut.

The next case I had was of the type described by Dr. Samuels, having gone to the stage of bone formation. The whole choroid was a lace-work of calcified material and the specimen formed here maintains the whole spherical shape of the choroid in the diagram of the eye, and there of course still remains stretched the remnants of the lens. That was a case of long duration.

These eyes are largely and for the most part shrunk. You can put a glass ball up to 20 mm. in size, in there, re-expanding them again to the capacity of the orbit. You then have a cornea that is really made of glass.

The astonishing thing to me was that the conjunctival epithelium would grow around the edge of the cornea and begin to line the inside of the sclera, between the sclera and the glass ball.

There has been no trouble with these cases. They give their eyes practically no attention. They move very well. This one does not move very well.

For a certain group of cases, which of course the pathologist will have to dictate — and it may be the ones where sympathetic ophthalmia is not present — very presentable results maye be obtained with this form of amputation, because practically all the sclera can be left. However, there will be large groups of tumor cases where it cannot be applied.

OCULAR PAPILLOMA

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Papillomata of the eye seems rare enough and presents so many interesting and perplexing features, that all cases with their most successful methods of treatment should be reported. One has but to search the literature and review the standard text books to appreciate the scarcity of published information, the limited number of reported cases and the absence of a standard or accepted treatment.

The vast majority of cases reported have been on the caruncle and palpebral conjunctiva but my report is limited to those appearing on ocular conjunctiva and cornea.

The etiology is an unsettled question. Some ascribe them to trauma, as an injury to the eye, or following foreign bodies as sand, cement, lime burns, and the removal of pterygiums.

I am convinced that, adult papillomata are due to trauma from wind containing much dust and grit as all cases seen were in a section where dust laden wind is prevalent and among those people who spent most of their time out of doors exposed to the wind. Furthermore all cases were on developed or potential pterygium which are caused entirely by exposure.

For papilloma in the juvenile we must find some other cause. The absence of any history of trauma or irritation suggests that there may be a congenital predisposition, and the rapid recurrence of papilloma following repeated removals might suggest that these growths are locally infectious. The apparent contagiousness of laryngeal papilloma and its infectious nature in animals might suggest a possible relationship to ocular papillomata.

Papillomata usually occur in one or other of two forms, diffuse papillomatosis, the villous mass with or without a pedicle, and the mushroom, cauliflower, or raspberry type. If the epithelial elements predominate, the papillae are closely packed and the growth is smooth. If the vascularized connective tissue is in evidence the

tumor has more branching processes and represents the typical appearance of a papilloma.

The pedunculated type has to be distinguished from granuloma, tubercle, vernal conjunctivitis, and condyloma, while the sessile type must be distinguished from carcinoma, lymphangioma, vernal conjunctivitis, xerosis, epithelial plaques and pterygium.

The limbus seems to be a common site for papillomata, because as Fuchs says, it is the only part of the ocular conjunctiva which normally contains small papillae.

My experience does not confirm the usual text book statement that locations of election are the plicae and caruncle even though the caruncle is a veritable pathological emporium, neither does my experience indicate that malignant degeneration is very common. In spite of the many common ailments and tumorous growths of the conjunctiva, true papillomata are relatively rare.

This report includes only cases of ocular papillomata clinically diagnosed and confirmed by pathological examination. It includes eight cases that were cured and one that resulted in enucleation.

Dr. E. H. Cary[1] of Dallas, Texas, reported the case of a man age 82 years. The condition was of one year duration and so extensive that a diagnosis of epithelioma was made and the eye enucleated. The pathological examination proved it to be only simple papilloma.

Dr. Martha B. Lyon,[2] of South Bend, Indiana, reported the case of a child 5½ years of age. This tumor was of two years' duration and supposedly followed an injury. There were four recurrences.

Dr. Robert J. Masters in discussing this case reported the case of a girl age 20 years, treated and cured by repeated applications of the thermophore.

Freytag[9] reviewed the literature reporting 34 cases, 11 of which occurred at the limbus and 6 on the ocular conjunctiva.

Dr. William B. Daugherty[3] of New York reviewed the literature and reported two cases. He stated, "In reviewing the literature I was surprised indeed to learn of the number of cases of papilloma of the cornea and corneo-scleral margin that presented both clinically and microscopically all the characteristics of these new growths and later developed malignant degeneration." In this respect I am more surprised than he, as none of my cases showed any malignant degeneration.

Dr. Julius Fejer,[4] Budapest, Hungary, reported a case of papilloma with diagnosis of an associated carcinoma due to a markedly atypical proliferation of the epithelium.

He states, "To establish a differential diagnosis between papilloma and carcinoma, is often rather difficult, although the first never penetrates into the depth of the corneal tissue, has a cauliflower or raspberry appearance and undergoes fungoid growth across the limbus without blending with the latter."

R. Beatson Hird[5] of Birmingham, England, reported three cases, one in 1913 of the caruncle, consulted for a chronic discharge of the eye. The growth was removed surgically and its base cauterized.

He was consulted by the second case, a boy of 14 years, in 1929 for a severe ocular hemorrhage. A large papilloma was removed from the inside of the lower lid. The third case was that of a woman age 39 with papilloma in the lachrymal sac. She had had repeated operations during childhood.

Dr. W. C. Souter,[6] of Aberdeen, Scotland, reported a case of diffuse papillomatosis in a plasterer, age 49 years, with a spontaneous cure. He reviewed the literature analyzing 32 cases.

Dr. M. F. Weymann[7] of Los Angeles, California, reported two cases, producing cures by the use of the Shahan Thermophore.

The first pathological description of a papilloma was made by Gayet[8] in 1879.

Practically all pathological reports define the growths as being chiefly fibrous tissue with groups of squamous epithelial cells. Groups of large and small cells with some fibrous vascular tissue and even those appearing at the limbus which receive their epithelial elements from the cornea, derive their vascular connective tissue core from the conjunctiva.

CASE REPORTS

(1) Mrs. A., a ranch woman, age 35, noticed a growth on the nasal side of the left eye some 8 months prior to consultation. It had grown rapidly since, with some tingling and shooting pains until it seemed to interfere with proper closure of the eye. The growth, 6 mm. by 10 mm., was removed and base cauterized. It recurred in two weeks when a cautery again was used and a cure secured.

FIG. 1 — (Windham) — Case II — Right eye, nasal.

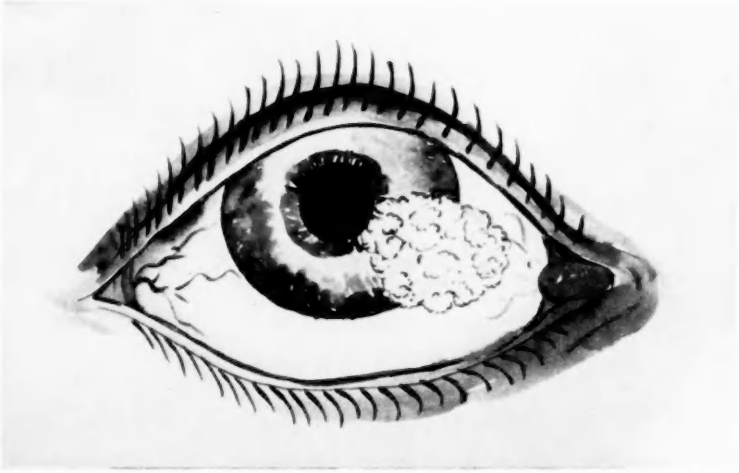

FIG. 2 — (Windham) — Case IV — Right eye, nasal.

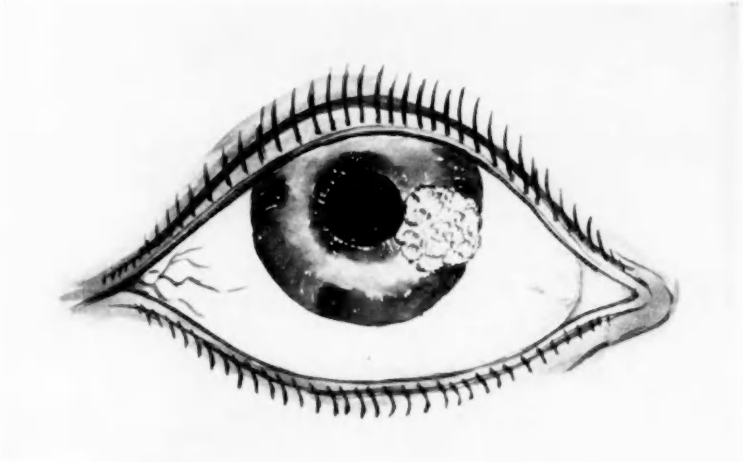

FIG. 3 — (Windham) — Case V — Right eye, nasal.

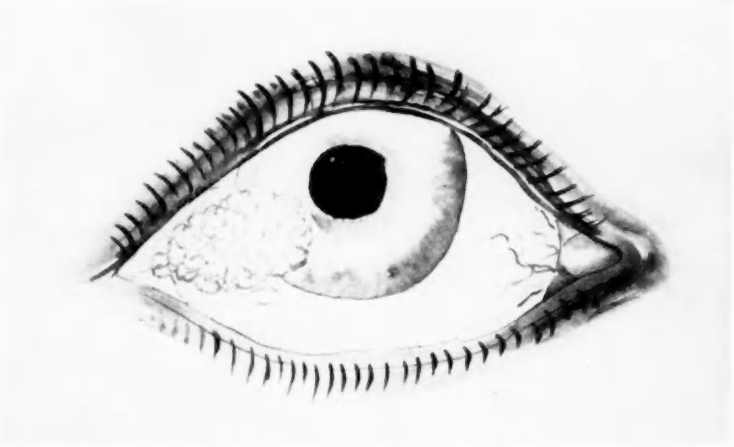

FIG. 4 — (Windham) — Case VI — Right eye, temporal.

FIG. 5 — (Windham) — Case VI — Right eye, below cornea.

(2) Mrs. M., age 30 years, wife of a railroad man. The growth, 10 mm. by 15 mm., was on the nasal side of the right eye and of 4 months' duration. It was removed and cauterized and cured in one treatment.

(3) Mr. S., age 40 years, a railroad mechanic, presented a tumor of the conjunctiva on the temporal side of the left eye, 8 mm. by 12 mm. of 3 months' duration. It was cured by one application of the thermophore.

(4) Mr. P., age 42 years, a ranchman, presented a tumor, 7 mm. by 9 mm., on nasal side of right eye at the limbus, the growth being of 6 months duration. It was removed and cautery applied. It recurred but was cured by application of the thermophore.

(5) Mr. W., age 65 years, a ranchman, presented tumors of both eyes, situated on the nasal sides near the corneas, and could be seen by the patient. The growths were of about 7 months' duration. Thermophore applied to each eye affected a cure with one treatment. The growths were about 5 mm. by 7 mm. in size.

(6) Mr. E., age 65 years, a rancher-farmer, applied for treatment for a large growth on the temporal side of the right eye, which was conjunctivo-corneal. He had been told by a doctor that it was cancer. The mass was extensive, fungoid or cauliflower in appearance and of distinct grayish color, extending down to the sclera and through Bowman's membrane. With local and ciliary

ganglion block anesthesia, the mass was removed and the base cauterized. The growth reappeared in the lower quadrant and was again removed and the base cauterized. Soon the tumor reappeared on the nasal side of the eye and again removed with cauterization of the base. The patient was not seen for several months later when he had a very large mass in the lower quadrant, pushing the cornea completely beneath the upper lid. At this time an orbital evisceration was done, curing a papilloma but sacrificing an eye. In spite of repeated operations and office treatments, this case showed no evidence of malignant degeneration.

(7) Mr. P., age 55 years, a traveling salesman, had a 5 mm. by 9 mm. growth on the nasal side of the right eye of one year duration. A single application of a thermophore resulted in a cure.

(8) Mr. L., a farmer, age 50 years, had a limbal growth, 6 mm. by 8 mm., of about 9 months' duration on the nasal side of the right eye. Thermophore application resulted in cure in one treatment.

(9) Mr. M., a laborer. This case was picked up accidentally while making a routine examination, January, 1937. A small growth, pinhead in size, was detected on a well developed pterygium on the nasal side of the left eye in the conjunctiva. This growth was diagnosed clinically as a papilloma and after explaining its significance to the patient, I agreed to remove the growth and pterygium free of charge if he would let me observe its growth and study it. This he agreed to, and I considered it a unique and interesting privilege to watch a papilloma grow from infancy to large size as the two pictures indicate. The growth was removed in May, 1938, approximately seventeen months after its first detection and at the time of its removal, it had grown from 1 mm. to 8 mm. in diameter. It was removed by one application of the Shahan Thermophore following which the pterygium was removed and to date there is no evidence of recurrence.

SUMMARY

(1) The etiological factor of papilloma is believed to be chronic irritation, as from dust laden wind, as all ocular papillomata are in the palpebral opening on pytergium or potential pterygium and those on the cornea are likewise in the palpebral fissure.

(2) Any case showing intra-ocular extension as in the anterior chamber, should be considered as a carcinoma, as the progression of a papilloma is forward and not backward.

(3) If papillomata are not due to irritation and exposure, no one has been able to explain why they occur only in the palpebral opening and not around the cornea.

(4) Whether due to trauma or to an invisable filtrable virus as suggested by Sir St. Clair Thomson, their tendency is to recurr unless killed at the initial treatment.

(5) Papillomata of the pedicled or loosely attached type are much more likely to be cured in the initial treatment, whereas the sessile type are less amenable to one treatment.

(6) Any case that shows no recurrence in the original area within one year should be considered cured and a recurrence after one year in another area should be considered to have come from the same cause as the original papilloma. A malignant lesion appearing after one year should be considered to have no relation to the original papilloma. These benign tumors are believed not to become malignant, and if malignancy is present, that condition is believed to exist from the beginning and not a change in the character of the papilloma.

(7) A papilloma begins as a small tumor or nodule with a characteristic appearance, then bursting into a lobulated cauliflower mass grows rapidly the first six or eight months. It then slows down into a rather sedentary cauliflower tumor of many lobulations with vascular tissue proliferation.

TREATMENT

(1) Various types of treatments have been tried. Radiotherapy should not be used because it seems to stimulate activity. Also it affects normal vital tissue too severely and leaves the cornea with a denser vascularized cloudiness than before.

(2) Careful resection of the papilloma and cautery of the denuded base will result in a fair number of cures. With the cautery one can control fairly accurately the degree of heat penetration, thus saving vital and uninvolved tissues.

(3) Diathermy may be used but one cannot accurately control its extent or penetration in order to avoid unnecessary tissue destruction.

(4) Even though Shahan (personal communication) does not especially recommend it for treatment of papilloma, the Shahan Thermophore should be the method of choice since papillomatous tissue in the eye melts away as if by magic at 150 to 160 degrees F., which is not sufficient to destroy normal tissue. With it one can melt away the growth, leaving normal, healthy, uncauterized tissue beneath and by careful, diligent application of one to two minutes, one may usually affect a cure with one treatment without scarring. Carcinomatous tissue will not melt away with this degree of heat.

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DISCUSSION

Dr. R. J. Masters, Indianapolis, Ind.: Having accepted the assignment of opening the discussion of this paper, I was very promptly confronted by the realization that I was not at all sure that I know what constitutes an ocular papilloma. Now, after careful study regarding this type of tumor, I cannot be sure that I have ever seen one, although I think that I have treated two of the sessile type. I have not encountered a papilloma of the lobulated, mulberry-shaped type, although it seems that this variety should offer less difficulty in its clinical diagnosis than the sessile type. Even in the microscopic section, a flat papilloma of the bulbar conjunctiva may be hard to distinguish from an epithelioma of low-grade malignancy. Clinically, several kinds of epibulbar tumors may look very much alike, and resemble a papilloma of the sessile type. To illustrate this point, I have chosen the following case histories for brief presentation.

Case 1. White female, age 16 months. A flat, smooth-surfaced, yellow-pink tumor of the bulbar conjunctiva of the left eye, extended from the semilunar fold outward and upward and downward so that the entire nasal half of the conjunctival surface was affected. Two treatments with the Shahan thermophore were employed, under general anaesthesia, two weeks apart. At the first treatment there were three one minute over-

lapping applications of a large tip at 150° F., covering the upper half of the tumor. The second treatment was similarly applied to the lower half. The tumor disappeared and did not recur. Two other ophthalmologists agreed with my tentative diagnosis of unpigmented naevus in this case. Far too large for excision, the tumor was never examined under the microscope.

Case 2. A white male, age forty-two years, was examined one month after his left eyeball received a glancing blow by a pipe wrench. Two days after the injury, a blister appeared on the bulbar conjunctiva immediately adjacent to the temporal limbus. After seven days the blister had changed to a firm pinkish-grey, flat tumor of oval shape, measuring 6.5X5 mm. Dilated vessels entered it from above, below and temporally. There was no infiltration of the cornea. The Shahan thermophore at 145° F. was applied for one minute. Two weeks later Dr. Shahan, visiting in Indianapolis, saw the tumor, which had reduced in size to 5X4 mm., and advised its excision. The pathologist reported that the sectioned tumor showed a small bit of squamous epithelium and granulation tissue overlying loosely organized connective tissue, with all of the underlying tissues presenting the features of a proliferative inflammatory process. This tumor was probably a granuloma, although its surface seemed to be fully and smoothly covered by epithelium. As small and firm as it was, it should have been excised in the first place. As the patient objected to excision the thermophore was used, which brought about some reduction in size of the tumor.

Case 3. White male, age 68 years. Six weeks following the subconjunctival injection of ten minims of 1:6,000 cyanide of mercury solution in the upper temporal quadrant of the right globe, a small elevation was noted at the temporal limbus. This tumor measured 5X3 mm., was of grey-pink color and had a small white ulcerated patch upon its surface. It overlapped the limbus, but did not infiltrate the cornea. Engorged conjunctival vessels entered it. Three months passed before this tumor was again observed. It had overlapped the cornea still farther, without involvement of the corneal tissue, and there was no ulceration of its surface. The 7 mm. thermophore tip at 158 F°., was applied twice for one minute, covering the tumor surface thoroughly. The tumor disappeared leaving the eyeball of normal appearance thirty months later. Early ulceration of the surface of this tumor suggested epithelioma. Subsequent healing of this surface ulceration, together with very slow growth and lack of corneal infiltration, suggest the possibility of a more benign tumor which followed the injection of mercury solution under the conjunctiva.

Case 4. White female, age 19 years. Four months before coming for examination she had noticed a fleshy growth on the nasal aspect of the left globe. Later a second growth appeared on this eyeball, and two similar lesions on the right globe. Examination revealed two grey-pink sessile tumors in the right bulbar conjunctiva, extending upward and downward, respectively, from the semilunar fold. Upon the left eye were two similar growths in similar position, the upper one being pedunculated. The latter was excised, the other three treated successfully with the

thermophore. There had been no recurrence seventeen months later. Section of the excised growth led to a diagnosis by the pathologist of epithelioma of low-grade malignancy. His detailed description, however, strongly suggested papilloma. There was a very vascular core, surrounded by a small amount of fibrous tissue containing many lymphocytes, with a covering of several layers of squamous epithelial cells which infiltrated the underlying structures at some points. This case was interesting and satisfactory, in that one growth was available for section while the others responded to thermophore therapy. The cause of the condition was subject to conjecture, but the patient had used mascara on her eyelashes in profuse amounts for many months.

I am glad that the essayist has again directed our attention to the use of the Shahan thermophore in the treatment of some epibulbar tumors. Soft flat growths which are not thicker than 1 mm. and which involve a large area of the bulbar conjunctiva, may be given a therapeutic test with the thermophore. Firm growths and those of moderate size should be excised. Papillomata which arise at the limbus, whether the type that grows outward from the surface, or the kind which invades the corneal substance, should preferably be excised. This applies probably to all of the pedunculated papillomata. There are, however, occasions when surgery is strongly resisted by the patient and another type of treatment is desired. The essayist has therefore performed a service to us by recounting his encouraging experiences with the thermophore in his large group of cases of papilloma of the bulbar conjunctiva. My less extensive experience supports his conclusions regarding the value of the instrument.

Dr. Wiley R. Buffington, New Orleans, La.: In the first place it must be conceded from statistical data that malignant or recurring ocular papilloma is relatively rare, in spite of the fact that Shumway, before the Section on Opthalmology, A.M.A., 1903, states that it is a common type of conjunctival tumor. Elschnig as early as 1889 said that they are rare. A review of data from six German authorities during the period from 1884 to 1899 suggests that papilloma is rather common.

To determine the exact frequency careful clinical diagnoses should be made; more important still, precise histological study. In a review of many case reports, I am inclined to think the latter has not always been thoroughly done. Granuloma arising over site of ocular muscle operations has been classified by one text book under the papilloma group; yet, these tumor-like formations are made up of inflammatory tissue. They do not recur after excision.

The case reports so well given by Dr. Windham show that recurring or malignant papilloma is far more frequent in southwest Texas than it is in Louisiana. In that section the climate is dry, the wind is heavily laden with dust and sand; in Louisiana, moisture free from dust fills the surrounding atmosphere. It would seem that conjunctival irritation from exposure of such substances is a most important etiological factor.

My statistical reports show that recurring papilloma is extremely rare in Louisiana. From 1922 to 1937, 14,686 eye patients were admitted to

Charity hospital for operations on the eye. Among these only eleven were authentic cases of ocular papilloma. In my private series only three cases are found among upward of 25,000 patients. These facts would suggest that climatic conditions may account for the relative infrequency of conjunctival papilloma.

True malignant recurring papilloma must be distinguished from other ocular conjunctival growths, hyperplasias, and conditions. Dr. Windham has named most of these. Papilloma has certain characteristics which distinguish it from other ocular conjunctival tumors:

1. Papilloma is a small flat tumor.
2. It has an uneven or velvety surface due to the fact it is covered by minute elevations. It is composed of branching papillae of connective tissue, surrounded by thick layer of stratified epithelial cells. The thin walled blood vessels often resemble endothelial tubes.
3. Conjunctival papilloma invariably recurs unless properly treated. In some cases it recurs again and again in spite of the most efficient treatment.
4. True papilloma never metastasizes.
5. It may undergo malignant or carcinomatous degeneration.

"The regular disposition of the cells, the intact basement membrane, and the delicate fibro-vascular case, help to indicate the difference between papilloma and carcinoma." (Souter.) The demarcation between malignancy and non-malignancy is not always easy to determine. Breaks in the basement membrane; invasion of tumor cells, suggest malignancy. Coover's case (A.J.O., V. 3, p. 683, 1920) is worth referring to. (See slide.)

This papilloma occurred in a scar at the temporal limbus in 1903. It was removed. The pathologic report was papilloma. In 1913 there was recurrence of a massive growth which was removed. Report was the pathologic basal cell epithelioma.

Gourfein (Review gen. d'Ophth. V. 42, p. 5, 1927) reports a case of epibulbar papilloma arising from a limbal scar.

Diagnosis: Papilloma undergoing carcinomatous changes. Pascheff, (Royal London Ophthal. Report XVI, 1905) states:

1. Primary limbal papilloma is rare.
2. It may develop quickly.
3. It recurs frequently after operation. Recurrence may occur in a few days or be delayed more than two years.
4. It often undergoes malignant degeneration.

Terrein and Cousin (Arch. d'Opht., V. 48, p. 622, 1931) discuss the close relation between limbal papilloma and carcinoma. In their opinion, papilloma may be the transition state of malignancy.

Preponderence of evidence from many other authorities confirms the reports and opinions of those just quoted.

Treatment: From a clinical standpoint, prompt and effective treatment is important to prevent recurrence and to forestall malginant degeneration.

Complete removal is not sufficient in the successful treatment of the conjunctival papilloma involving the cornea. After removal the denuded

area must be treated by some cauterizing agent. Recurrence may take place in the original site. Usually, however, if the tumors are astride the limbus the recurrence is an extension above or below. Frequent observation of the lesion is important. With the first sign of recurrence, prompt treatment must be instituted. This may be once, twice, or many times. Removal and cauterization are more effective in the treatment of papilloma than irradiation, or even combined removal and irradiation. The cauterizing agent may be the electric cautery, the thermaphore, or a chemical such as glacial trichloracetic acid. In my hands the latter has been the most efficient. It can be used with great frequency with a minimum permanent damage to the cornea and sclera.

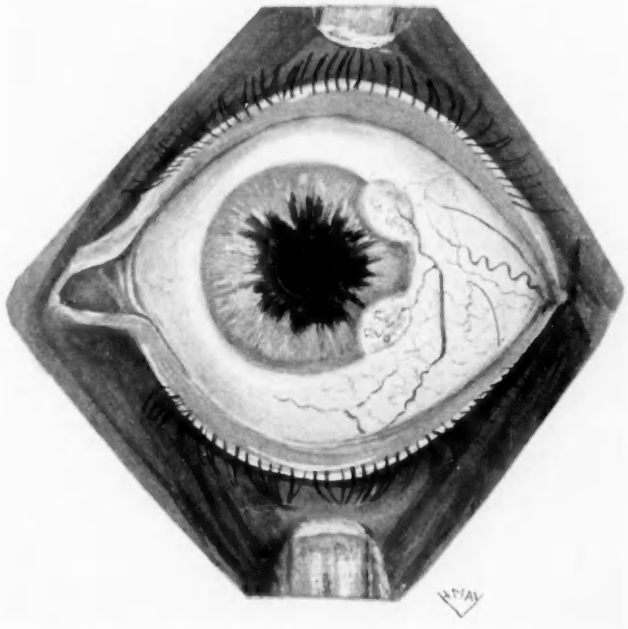

FIG. 1 — (Buffington)

CASE REPORTS — OCULAR PAPILLOMA

Case One. On June 14, 1932, Mr. F. T. L., who was 44 years of age, white, presented a recurring growth on the conjunctiva and limbus of the left eye. It had been removed four times since August, 1931, the last operation having been performed on May 1, 1932. Examination of the left eye revealed two flat vascularized tumors involving the conjunctiva and cornea (Fig. 1). X-ray and radium treatments had been applied by Doctor V. from June to December, 1932. On December 15, 1932, the tumors, in spite of x-ray and radium treatment, had grown steadily and became confluent

at the temporal limbus (Fig. 2). On December 17, 1932, the tumors were removed under local anesthesia. They were found to be attached to both the sclera and the cornea. The denuded surfaces were treated with electric cautery, and the area covered with a sliding conjunctival flap. Two small recurrences at the upper limbus were removed and cauterized on May 18, 1933. On May 24, 1933, a 5 mm. round, flat tumor astride the upper limbus had returned. It was removed and the surface was treated with electric cautery. On January 8, 1935, recurrence of the growth along the temporal limbus was noted. This time it involved half the temporal limbus (Fig. 3).

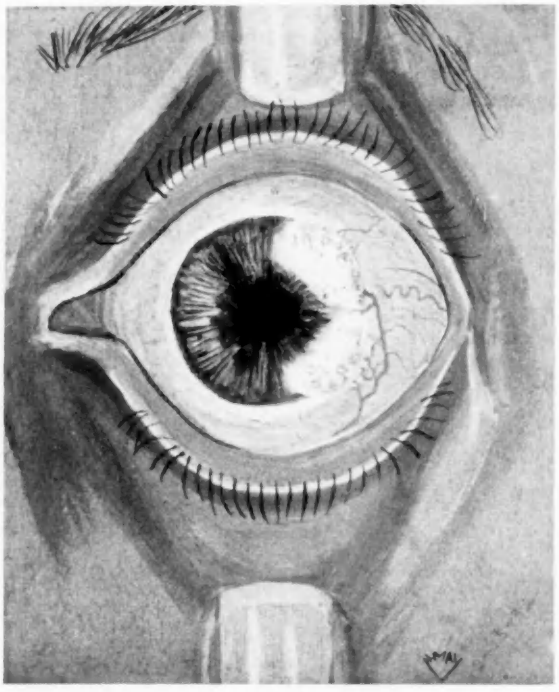

FIG. 2 — (Buffington)

Recurrence was noted by the patient in one week. On January 22, 1935, I completely excised the papillomatous growth. The attachment of this recurrence was at the limbus. The tumor overlapped the cornea. The whole denuded surface was then touched with glacial trichloracetic acid. The clinical diagnosis was recurring papilloma of the conjunctiva and cornea, and the pathologist's report was papilloma, suggestive of undergoing a squamous-cell carcinomatous degeneration. To date there is no recurrence. Vision of both eyes is 20/20, or normal (Fig. 4).

Case Two. On January 17, 1936, Mrs. G. A., who was 58 years of age, white, was seen with a growth on the right cornea, pin-head in size, first noticed about six months previous to this date. Prior to the appearance of the growth, the eye was red. The growth doubled in size in three months. The tumor was flat, vascularized, overlapping the cornea about 2 mm. There were prolongations along the limbus downward. The tumor was loosely attached to the conjunctiva and cornea (Fig. 5). It was removed, and the base touched with glacial trichloracetic acid. Clinical diagnosis was papilloma of the conjunctiva and cornea; pathologic diagnosis was papilloma, no

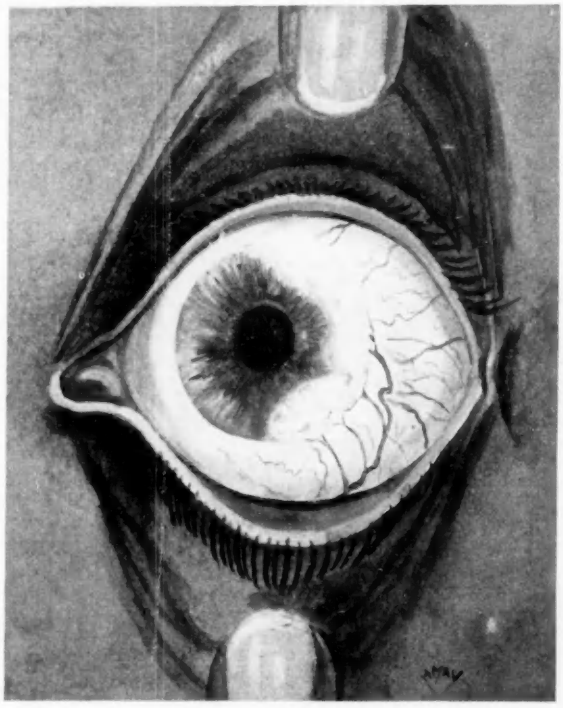

FIG. 3 — (Buffington)

malignancy. A recurrence of the papilloma was noticed on October 15, 1936, extending from 5 to 9 o'clock along the limbus. It was removed and the whole area touched with trichloracetic acid. On May 5, 1937, a recurrent, flat, 4 mm. papilloma came at the lower limbus. A smaller recurrence was noted at the upper limbus. Both were treated by excision and trichloracetic acid. There has been no recurrence to date. Vision, October 1, 1938: right eye, 20/40, left eye, 20/20.

Case Three. Mrs. A. C. C., age 29, white, was seen on November 11, 1936, because of a growth on the right eye of four months' duration. A 5 mm., round, circumscribed tumor of the conjunctiva was situated nasal to the limbus. The surface was rough, and leading into the mass were several large blood vessels. A diagnosis was made of papilloma of the conjunctiva. The tumor was removed, and the denuded area touched with glacial trichloracetic acid, and then covered with conjunctiva. The pathological report was squamous cell carcinoma. There has been no recurrence.

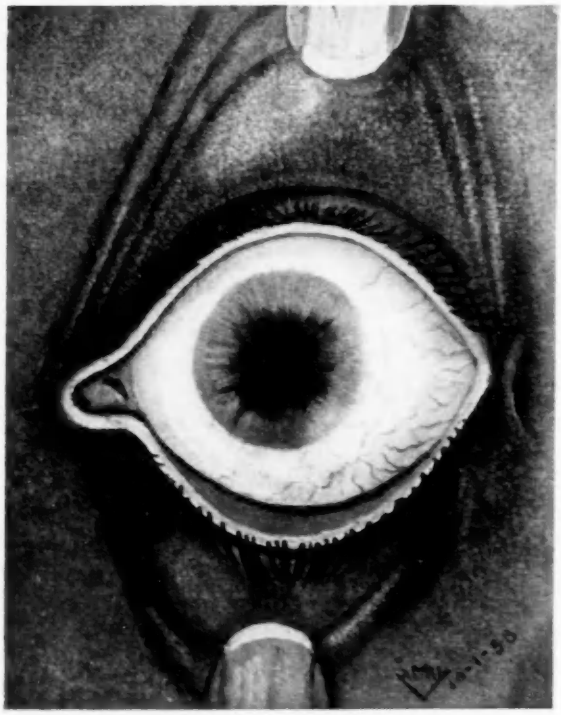

FIG. 4 — (Buffington)

FIG. 5 — (Buffington)

DR. EDWARD STIEREN, Pittsburgh, Pa., illustrated with lantern slide a huge cauliflower growth of the right bulbus in a male, age 45. Arising over the external rectus by a strong pedicle, it took on a mushroom shape, and was 12 mm. in length, 8 mm. in width, and about 10 mm. high. It reached to the external canthus, overlapped the cornea to its center and projected forward between the lids, which could not be closed over it. The surrounding conjunctiva was inflammed and thickened and there was considerable ropy discharge. The color and general appearance of the mass was that of a pale red raspberry. There was no involvement of the preauricular or any of the lymphatic glands. According to the patient's statement the lesion was first noticed about a year previously as a localized inflammation with a slight elevation.

The growth was excised and the defect covered with a sliding conjunctival flap. At only one point, over the insertion of the external rectus, was it adherent to the globe. Healing was prompt and uneventful. The specimen was pronounced to be an epithelioma of papillomatous type or a

papilloma taking on epitheliomatous changes. He was given X-ray exposures at intervals over a period of six weeks and when seen a year later the entire conjunctiva was smooth and glistening with no semblance of a recurrence.

Dr. Norman W. Price, Niagara Falls, N. Y.: Mr. R. J. came to my office August 12, 1937, with a growth on the 9 o'clock line at the limbus of his left eye. This was a flat growth extending a short distance over the cornea. I removed the growth with a cataract knife. The growth was only slightly raised and contained no pedicle. It was sent to the laboratory at Memorial Hospital, Niagara Falls, and also to Gratwick Laboratory, Buffalo. They reported the growth a papilloma.

Patient returned in two weeks with a growth quite as large as before. I then took him to the Gratwick Cancer Laboratory. They tried to remove the growth with an electric cautery on different occasions but it returned soon after each treatment. On Novmeber 10, 1937, growth was very much in evidence.

March 7, 1938, at the hospital I removed the growth thoroughly with a knife and cauterized the base thoroughly with trichloracetic acid, covering the area with a conjunctival flap. Another biopsy at this time showed a papilloma.

A month later growth was quite as large as ever and as Gratwick refused to use radium I did a thorough job of cauterizing, burning the tissues all I thought the eye could stand and again covering with conjunctiva the best I could.

Since then there has been no recurrence.

Vision corrected is 20/30.

Dr. Lawrence T. Post, St. Louis, Mo.: I wish to express agreement with the essayist in the value of the thermophore in the treatment of tumors of the type described. In our office we have been using the thermophore in these cases, and also in the clinic at Washington University for the past twenty years. We have had excellent success and have had very few recurrences. I believe the reason one has fewer recurrences in treating with the thermophore is because heat from the thermophore penetrates further than other types of cauterization, though as the essayist said, penetration probably is limited to about a millimeter and a half, or thereabouts.

I would take exception to his statement about malignant growths. We have found that they were equally successfully treated by the thermophore. We find that depending upon the condition of the tissue with regard to its relationship to scar tissue, it tends to be unsuccessful. The softer, the newer the growth, the more successful is the thermophore application.

About three years ago an essay or thesis before the American Ophthalmological Society pointed out that the degree of heat did not register accurately in a series of thermophores which the author tested. Dr. Shahan recently has gone over this and found that statement to be correct. He has

recently devised a new thermophore which is far more accurate. I think that a number of failures in thermophore therapy in its various applications may have been because of inaccuracy of the thermometer in the instrument. It is possible to standardize your own thermophore by utilizing the fact that the melting points of drugs are relatively critical, within the matter of a degree or more. For example, the melting point of trional is given as 168.8 degrees Fahrenheit. If you place a few crystals on the contact surface of your thermophore and set it for that temperature, you will find that though melting point and the re-crystallization are within one or two degrees, it may not read 168.8 on your thermophore. Therefore you can by such a method — and there are other drugs for other temperatures — determine with fair accuracy the error in your own thermophore. We hope that the new one will be far more accurate.

I congratulate the essayist on the success that he has had. I might say the temperature we have used is 150 degrees for one minute, remembering always that it is necessary to make continual contact during that minute.

DR. R. E. WINDHAM, in closing: I wish to express my deep appreciation to all the discussors for the very interesting and, to me, enlightening remarks that they have made.

I first used the thermophore on these growths experimentally. I had a case of one of the smaller groups, I think it was the third case I had, and I used the thermophore merely to see what would happen. I used a tip larger than the growth and held it for one minute by the watch, raised my tip to see what had happened to the tumor and found I didn't have a vestige of it left. I thought I had found something. Thank you very much.

THE PRACTICAL USE OF HOMATROPIN-BENZEDRINE CYCLOPLEGIA A FURTHER REPORT*

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STIMULATED by the work of Myerson and Thau[1] and recent reports of Beach and McAdams[2] regarding the use of Benzedrine Sulphate (Amphetamine Sulfate) as an adjuvant in cycloplegia, a number of studies have been undertaken using some four adrenergic drugs, viz., Adrenalin, Benzedrine, Ephedrine and Paredrine, in conjunction with Homatropine for the production of practical cycloplegia in the refraction of young adults. The Homatropin - Benzedrine combination has seemed most satisfactory.[3,4,5] It is felt that cycloplegia is necessary in the routine ophthalmological examination and refraction of the average young adult. This is necessary, of course, both that the refraction may be more accurately estimated and also that the interior of the eye may be more completely and more satisfactorily examined. While the orthodox Homatropine cycloplegia has proven quite satisfactory from the standpoint of the eye physician, still from the standpoint of the patient some objections — discomfort, annoyance and loss of time — are rightly offered. Certainly cycloplegia as complete as the usual Homatropine procedure but with much shorter duration and correspondingly less discomfort and annoyance is desirable. In the case of students, business men, stenographers and others of like vocations, the inability to do close work for a period of eighteen to twenty-four hours may be a problem of major importance.

At the Osawatomie State Hospital physically sound, co-operative patients, both male and female, of all age groups, are readily available for study. Following the studies among these groups, collateral use of these cycloplegic procedures has been carried out in the author's private practice. In determining the state of cyclo-

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plegia the patients were refracted first by retinoscopy with fixation in the distance. This was compared with retinoscopic findings with fixation at 30 inches. Marked discrepancy in these findings indicate lack of complete cycloplegia. Next, the trial case refraction for distance was determined and with this before the patient accommodation was checked with the Prince rule. And finally, following the suggestion of Dr. Rutherford[6] the degree of cycloplegia was estimated by placing a plus 3.00 D sphere before the distance correction and checking the far point at 33 centimeters.

The usual Homatropine cycloplegic procedure was first studied and calibrated. For this group 24 patients within the 16 to 31-year range were studied. The following observations were first made:

1. The size of the pupils in millimeters.
2. The reaction of the pupils to light and accommodation.
3. Distant vision as determined by the Snellen chart.
4. The ability to read Jaeger test type.
5. Accommodation as measured by the Prince rule.

Patients exhibiting any pathological variations from the normal were excluded. Homatropine Hydrobromide aqueous solution 2% was instilled in each conjunctival sac at the outer canthus every five minutes until 4 instillations were given. Observation of the above named details were made at the ½ hour, 1 hour, 2 hour, 4 hour, 8 hour and 18 hour intervals. Complete practical cycloplegia occurred in the majority of cases 1 hour after instillation. A beginning recovery of accommodation was observed in about half the cases 8 hours after instillation. Complete recovery of accommodation as judged by the ability to read Jaeger 1 type did not occur in the vast majority of instances until or after the 18-hour interval. The pupillary size increased an average of 4 mm., reaching its maximum ½ hour after the last instillation. Beginning diminution in the size of the pupil did not occur until the 8-hour interval and had not regained the normal size at the end of 18 hours.

In studying the Benzedrine Homatropine cycloplegic reaction, similar groups of both male and female patients were selected, ranging from 16 to 31 years of age. One hundred cases were studied. Various modifications of technique were used experimentally. Two instillations of Homatropine 2% solution were found to be more uniformly effective than one drop of a 5% solution. Likewise, two instillations of one drop each of the 1%

Benzedrine Sulfate ophthalmic solution produced a larger pupil and greater clearness of the cornea than a single administration. The following technique was finally adopted as most suitable. Two drops of Homatropine aqueous 2% solution was instilled in each conjunctival sac at the outer canthus 5 minutes apart, 2 instillations thus being given of 1 drop each. This was followed in 5 minutes by 2 similar instillations 5 minutes apart of 1% Benzedrine Sulfate ophthalmic solution. Beginning recession of accommodation was evident in ½ hour and at the end of 60 minutes complete practical cycloplegia existed in 93% of the cases. A beginning return of accommodation was evidenced by the ability to read Jaeger type at the end of 4 hours in about 50% of the cases. At the end of 8 hours 75% of the cases were able to read Jaeger 1 type and at the end of 18 hours there was a complete return of normal accommodation in all of the cases as measured by the Prince rule and the ability to read Jaeger 1 type. One-half hour after the first instillation the pupil in all cases had reached its maximum dilatation. The average dilatation was 4.5 mm. A beginning return of the pupil to normal was evident in most cases at the end of 4 hours and was complete at the end of 18 hours, in all cases. It was noted that recovery of accommodation occurred before the normal pupillary size was finally attained.

The adrenergic action of the Benzedrine seems to have a definite clarifying action on the cornea as well as increasing the dilatation of the pupil. Both these factors are definite aids in the use of the ophthalmoscope and retinoscope. While the Homatropin-Benzedrine method has been found eminently successful in young adults, it is believed that in children, whose amplitude of accommodation is so much greater, the "Homatropine alone" or "Atropine alone" procedure is to be preferred. As a general rule, children under school age are examined by the "Atropine alone" method while in those of school age the "Homatropine alone" method is used.

The action of miotics in bringing about a return of accommodation following Benzedrine-Homatropine cycloplegia has been studied in 24 cases and Eserine solutions have been found the most effective.[4] Eserine Salicylate solution buffered to a p_H of 6.2 making it isotonic with the tears has been used to advantage. A similar group was administered Benzedrine-Homatropine cycloplegia under the conditions described above. One and one-half hours following the administration of the Homatropine, ½ of

1% Eserine Salicylate buffered solution drops 1 was instilled in each conjunctival sac. Eserine Salicylate ½ of 1% buffered solution overcame this cycloplegia and brought about a practical return of accommodation ½ hour after its administration. This was followed by a moderate but definite diminution in accommodation. However, as the decline of the effect of Eserine progressed, it was met by the natural recovery from the effect of the drugs, so that the cycloplegia was overcome in all cases 5¼ hours following the administration of the first drop of Homatropine. The use of 1% buffered Eserine Salicylate solution also brought about a complete return of accommodation ½ hour after its administration, but this stronger solution exhibited a more lasting effect so that at the end of 4½ hours all patients were able to read J 1 type. No serious or untoward effects have been noted from the use of ½ of 1% buffered Eserine solution but in private practice a repeated dose of the 1% buffered Eserine Salicylate solution caused one patient to have a violent attack of nausea and vomiting.

The following is the record of a graduate student in the Department of Physiology at the University of Kansas just recently examined. This man was arbitrarily chosen before examination as a case for presentation because of his understanding of the problem and his accurate observations. He proved to be an excellent case for demonstration. Several interesting points are noted:

1. Loss of accommodation in 60 minutes.
2. Extreme dilatation of the pupil.
3. Beginning recovery of accommodation in 4 hours.
4. Progressive but still incomplete recovery of accommodation at the 8th hour. (As a general rule younger persons exhibit a more rapid and complete recovery due to their greater amplitude of accommodative power.)
5. The rapid return of accommodation following the administration of 1% buffered Eserine Salicylate solution.
6. The return of accommodation in advance of pupillary recovery.
7. The "come and go" effect on accommodation and pupillary size, of the Eserine as it struggles for supremacy of effect.

At the 8th hour Eserine Salicylate 1% buffered solution was administered and six minutes later he could read J 1 type with right eye, J ii with left eye. Fifteen minutes later or 21 minutes after the Eserine was administered, the accommodation had advanced to the normal amount in each eye, although he was able to

read only J iii. This seeming contradiction was probably due to the extreme contraction of the pupils and the confusion resulting from the conflict between the cycloplegia on the one hand, and the natural tendency to recovery tremendously stimulated by the Eserine solution on the other. Twenty minutes later the eyes had reached their normal state both as to accommodation and pupillary size, as well as the ability to read J 1 type. There were no subsequent variations.

MR. J. P., AGE 31 YEARS
OD — 075 = — 050 x 165
OS — 075 = — 050 x 15

0 hr.	½ hr.	1 hr.	2 hr.	4 hr.	8 hr.	8.06	8.21	8.41	10 hr.
J i	J ii	J o	J o	J vii	J vi	J 1	J iii	J i	J i
J i	J iii	J o	J o	J o	J vi	J ii	J iii	J i	J i
10 D	2.25 D	o	o	2.50 D	3.50 D	4.75 D	10 D	10 D	10 D
9 D	2.50 D	o	o	2.00 D	3.50 D	4.25 D	10 D	10 D	10 D
*3.5	8	9	9	9	8	8	1½	3.5	3.5
*3.5	8	9	9	9	8	8	1½	3.5	3.5
					Eserine 1% administered				

* Millimeters.

SUMMARY

Homatropine and Benzedrine in combination used according to the suggested procedure gave complete practical cycloplegia in a high percentage of patients at the end of 1 hour. The Homatropine-only groups showed a more rapid onset and a longer duration of cycloplegia with a subsequent delay in the return of accommodation. In the Homatropine-Benzedrine group there was a beginning return of accommodation at the end of 4 hours as contrasted with the Homatropine-alone group which shows a beginning return of accommodation in some cases only at the end of 8 hours. There was a tendency to greater dilatation of the pupil in the Homatropine-Benzedrine groups and the cornea was definitely clearer in these groups than in the Homatropine-only groups. One-half of 1% buffered Eserine Salicylate solution overcame the cycloplegia and brought about complete return of accommodation

½ hour after its administration. This action showed a definite tendency to wear off and was not as permanent or lasting as that observed following the administration of the 1% Eserine Salicylate buffered solution.

CONCLUSIONS

Homatropine and Benzedrine used in combination will produce in 60 minutes complete practical cycloplegia in a high percentage of patients between the ages of 16 and 31. Beginning recovery is evident in 4 hours. Eserine Salicylate solution 1% and ½ of 1% buffered to be isotonic with tears will overcome Homatropine-Benzedrine cycloplegia promptly and restore the power of accommodation within ½ hour.

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DISCUSSION

DR. C. W. RUTHERFORD, Indianapolis, Ind.: Studies have been made during the past year on the effects of benzedrine (amphetamine) and paredrine when added to homatropine for cycloplegia, and on the significance of variations in the location of the far point (Transactions of the American Academy of Ophthalmology and Otolaryngology, 1937, pp. 188-189). Both eyes of 174 private patients, including a few young myopic persons, were examined.

The patients were studied in six groups (Table I). All eyes received pontocaine to control blepharospasm, and then homatropine in amounts appropriate to the ages of the individual patients. Aqueous benzedrine sulfate, one drop of one per cent solution, was added to one eye of each patient in Group II and to both eyes in Groups III and VI. Paredrine hydrobromide, one drop of one per cent solution, was added to both eyes in

Group IV and to one eye in Group V. Group I, included for comparison, was tabulated from records made before the study was begun. Groups II to VI were consecutive.

No factors such as age, sex, occupation, character of ametropia, presbyopia and so forth were of value for predicting the probable location of the far point.

Percentages of far points are given in centimeters and diopters in Table II. Assuming that the test is a practical one and that a variation of 0.25 diopters from 33.3 cm. (between 30.7 and 36.4 cm.) is acceptably good refraction, then Group IV indicated that homatropine and paredrine were more strongly cycloplegic than homatropine alone, as in groups I and II; but Group V, in which the effects of the two methods could be compared in the two eyes of each patient, indicated that neither was materially stronger than the other.

Eyes in Groups I and II that had homatropine alone showed 12¼ per cent of far points outside the accepted variable of 0.25 diopter, and eyes in Groups II and III that had homatropine and benzedrine showed 11½ per cent outside. But of the eyes in Group V that had homatropine alone, all showed far points within the 0.25 D. variable, so it seemed unwise to discount the value of benzedrine without testing it again and with the more careful technic used in Group V. Accordingly, Group VI was studied.

Groups IV, V and VI showed all far points within the 0.25 D. variable, a conspicuous improvement over what was found in preceding groups. This was due to an awakened conviction that human variables were responsible for diopter variations, and that greater care would be necessary in conducting future tests.

Close attention to the behavior of patients was rewarded with interesting and instructive revelations. 1. The test should be made leisurely. It is advantageous to watch the patient's lips; as soon as they move, as though to speak, the test card is halted and then advanced very slowly until the designated word is read. 2. A daring patient attempts to read the word before it is clearly visible while a cautious one waits until sure of it. 3. Some persons can read words of square letters, like L E T, farther away than words of round letters, like C O D, while others can recognize round letters at the greater distance. Frequently a warning of this peculiarity will be detected while testing central acuity at six meters with letters of both forms in the same line, such as L C E O T D. 4. The test should be repeated until the operator is satisfied.

Conclusions: 1. The far point test is a practical one. A variation of 0.25 D. from 33.3 cm. (between 30.7 and 36.4 cm.) is an expression of variability in human behavior or aptitude and it cannot be eliminated; there can be no uniform precise location of the far point. 2. Benzedrine and paredrine did not augment the degree of cycloplegia obtained with homatropine alone; both increased mydriasis, which was of no apparent advantage; both shortened the duration of accommodative disability, which was a welcome convenience to patients.

The solutions of benzedrine and paredrine were obligingly supplied by Smith, Kline and French Laboratories, Philadelphia.

TABLE I

	Men	*Ages*	*Women*	*Ages*
Group I	13	20 to 53	12	18 to 53
Group II	25	17 to 48	15	19 to 51
Group III	13	18 to 55	19	11 to 53
Group IV	14	13 to 57	18	17 to 60
Group V	10	20 to 59	15	18 to 58
Group VI	7	11 to 50	13	21 to 60
Totals	82	11 to 59	92	11 to 60

TABLE II

Pr. in cm.		28.6	29.6	30.7	32.0	33.3	34.6	36.4
Pr. in D.		.50	.37	.25	.12	.00	.12	.25
Group	*Eyes*	*Pct.*	*Pct.*	*Pct.*	*Pct.*	*Pct.*	*Pct.*	*Pct.*
Group I	HA 50	8	10	8	14	38	12	10
Group II	HA 40	0	5	20	12½	32½	22½	7½
	HB 40	10	5	7½	17½	32½	25	2½
Group III	HB 64	3	6¼	23½	17	23½	25	1½
Group IV	HP 64			7¾	18¾	25	39	9½
Group V	HP 25			12	8	12	64	4
	HA 25			4	16	4	64	12
Group VI	HB 40			5	25	32½	32½	5

HA — Homatropine alone.
HB — Benzedrine added.
HP — Paredrine added.

DR. S. JUDD BEACH, Portland, Maine: It is very gratifying if the work that Dr. McAdams and I did in benzedrine and cycloplegics has been any factor in this very competent presentation of Dr. Powell's. Continuing the work that was reported by us at the American Ophthalmological Society and the Academy last year, we are rather impressed with the notion that single instillations of our ordinary cycloplegics are much more effective than we have in the past been accustomed to believe, and that is more or less consistent with the earlier use of cycloplegics which if you look back through the very beginnings of the literature, was to make single instillations of atropine. Now these single instillations of cycloplegics are so effective that it requires only a slight boost to make them sufficient for examinations of refraction, and this slight boost can be given in a number of different ways.

Our experience with these drugs rather leads us to believe that it doesn't make a tremendous lot of difference whether you use the con-

ventional method of repeating the instillation with the idea of getting cumulative action, or whether you use single instillations as Dr. Powell has described, reënforcing them with the adrenergic drugs, and also we are rather inclined to think that it makes relatively little difference whether you use the one per cent benzedrine or one per cent paredrine, or has been more recently recommended, paredrine in a 3 per cent solution. Also it seems to produce a very similar result if you combine the solutions. It is perfectly possible, as we mentioned in our early reports, to use the cycloplegic and the adrenergic drug in the same solution in the manner in which cocaine and homatropin have been customarily combined.

The results of these things rather indicate that it is largely a matter of preferential use and the way in which you use them. At first we were firmly convinced that one drug was distinctly better than another, but after we compared the results in the same eyes at different times, using different drugs, we found that the variation which at one time indicated that one drug was better, the next time might show that the other drug was quite superior, and paradoxically enough, sometimes we found in the same patient that a one per cent solution of paredrine, for instance, has apparently been more effective in the same combination with a cycloplegic than a three per cent solution, and this has been more or less confirmed, I think, by Tassman's experience with paredrine which he recently published.

We have rather inclined to try to make use of observations made on a few intelligent and cooperative patients than to attempt to get mass figures.

I like very much Dr. Powell's phrase "complete practical cycloplegia," because cycloplegia which we ordinarily get and which is quite satisfactory for the purposes of refraction is, as he said, very remote from complete cycloplegia.

As we have previously stated, we still are convinced that the effectiveness of the combination depends largely on proper instillation of the cycloplegic. We have found it necessary to be particularly careful about the use of the adrenergic drugs which seem to be quite effective. I think those who are using the strong solutions of adrenalin would probably agree with this. But a cycloplegic can very easily be flicked off or squeezed out of an eye and rendered almost inert. We still like our original routine which has been to instill two drops of the cycloplegic and one drop of benzedrine or paredrine, whatever is preferred, and we still prefer five per cent homatropin to the two per cent. There are a number of theoretical reasons why this should be more effective, and we are rather inclined to the idea that it is.

The point where our experience diverges from that just reported by Dr. Powell is in the use of homatropin alone and atropine alone for children. I have no reliance whatever on homatropin alone used in the conventional way for children of school age, and in fact for any patients under sixteen years of age; and conversely, I feel that one of the greatest benefits and comforts to patients that I have had has been in the combining of the adrenergic drugs with atropine for patients under sixteen years of age. It is quite a comfort to be able to make one instillation of these combinations in the office and have them work inside of an hour, and in school children to be reasonably sure if this is done on Friday afternoon that they are going to be able to go back to school on Monday without any difficulty.

Our results still confirm us in the findings of our original experiments, namely, that we get within about a quarter of a diopter of as good relaxation as we have obtained in the past by the three-day performance of the instillation of atropine alone, and of course with much greater comfort to the patient and to the family who don't have to do any of this job at home.

There is one thing that I think would be worth cautioning those who have never used this combination, and that is that benzedrine, and paredrine perhaps to a less degree, seem to dilate the tear duct very widely and very rapidly, so that in very rare instances but enough so that you have to be on the watch for it, you get quite marked and prompt flushing of the face following the atropine instillation. This does not, however, follow if you use the adrenergic drug after having made both instillations of the cycloplegic, and that may explain, possibly, that instance that Dr. Powell reported in which he got nausea and vomiting following eserin. Of course, if you use eserin after you have the tear passages widely dilated, and repeat it a few times, you do get considerable absorption of eserin.

We can certainly confirm the author's experience with regard to the use of eserin. The use of eserin after ordinary conventional use of homatropin, is followed by very prompt apparent recovery from the cycloplegic but the effect of the eserin soon wears off and the homatropin action returns. In cases, however, in which the action of the eserin is maintained approximately as long as the action of the cycloplegic, quick recovery is maintained.

Dr. William Crisp, Denver, Colo.: Nowadays I practically never have any trouble from rapid absorption of the cycloplegic drugs. The reason is that I almost invariably insist that the patient shall have a meal a short time before I do the instillation. I called attention to that some years ago. If the patient has something in his stomach, you will hardly ever get disturbance from the use of atropine and homatropin, and you are much less likely to have disturbance from the use of hyoscine in moderately sensitive patients.

The combination which I personally have been using since I learned of the experiences of Doctors Beach and McAdams has been as follows: I use, first of all, a two per cent solution of cocaine; four minutes later a one per cent solution of benzedrine or more recently, because the manufacturer seems to think that paredrine is a little better than benzedrine and is sending out the sample preferably of paredrine, one per cent of paredrine; then four minutes later one drop of 5 per cent homatropin. I am disposed to think that my experience has borne out Dr. Beach's statement that you get better results from one drop of the stronger solution of homatorpin than from the two drops of the weaker solution. I used to use two drops, two and a half per cent.

Now, this combination of mine which I have found very satisfactory, after one hour, meets the objection that has been raised to the use of the paredrine before the homatropin, namely, that you shrink the passage and tend to carry off your drug too rapidly. With the proper method of instillation I don't believe this objection has any particular validity.

We also put the drugs in above the cornea so that the film of solution at once spreads over the whole cornea. I believe that the presence of the

film over the cornea is the most essential part of the procedure. I think any excess of the drug which you may put in will not have much chance of absorption anyhow, because it either flows down onto the cheek or it goes into the lower culdesac and is rapidly taken care of by the nasal ducts.

I do believe that I gain materially as to the other two drugs by the preliminary instillation of the two per cent solution of cocaine, because not only do I anesthetize the patient's eye as to sensitiveness to the next instillation, but I have the well known further synergistic action of cocaine with the other drugs. My experience has quite borne out this line of argument. In fact, in some cases, even young people, it seemed to me I had perhaps as perfect cycloplegia as was ever obtained, as indicated in both the near point and far point test. The use of the term "far point test" seems rather confusing to me and I prefer to say as indicated by the extremely prompt reaction of the patient to very slight changes in the sphere, even sometimes objecting to a slight reduction in the total amount of plus sphere which I have worked out because it was said that the vision was not so good.

As to the use of eserin, I usually use one in 240, or as it is made up, one grain of eserin sulphate or salicylate to a half ounce of distilled water, and I have repeatedly observed that the patient's condition the same evening is better than that the next morning. Dr. Beach rather jocularly said the patient had hardly got out of your office before the effect of the eserin had worn off. Usually that evening the patient gets along pretty well but the next morning he is pretty apt to relate to you that he doesn't get along as well as he did the night before. That is because the effect of the eserin is diminishing very rapidly and the effect of the homatropin is still there to some extent.

I think this technic is quite an addition to the convenience of carrying on our work, both as regards the patient and as regards the work that has to be done by the office assistants.

Dr. I. S. Tassman, Philadelphia, Pa.: I began the use of benzedrine sulphate in aqueous solution in addition to homatropine hydrobromide following the work of Dr. Beach, and in the same manner I completed quite a number of cases and the results that I obtained correspond very closely with the results that were reported by Dr. Beach.

I began in using benzedrine sulphate to use the same strength solution that Dr. Beach reported, 5 per cent solution, and then experimented in various ways, using weaker strengths, including two drops of the two per cent solution in the way that was described by the essayist this afternoon.

About a year and a half ago the effect of paredrine came to my notice, after an investigation by two investigators at the University of Pennsylvania, I then substituted paredrine hydrobromide aqueous solution for the benzedrine sulphate solution, also trying various strengths, beginning with two drops of the two per cent solution, one drop of the four per cent solution, and again one drop of a five per cent solution. Both in the case of benzedrine and paredrine, I can't say that the results I obtained were as satisfactory with the two drops of a two per cent solution as those obtained with the one drop of a four per cent solution or a five per cent solution.

I did feel that the results with the one drop of a four per cent solution were practically the same as those I obtained with one drop of a five per cent solution.

I feel, as Dr. Beach does, that the important thing is the accuracy in the instillation. Where only one drop of a drug is being instilled into the eye, it must be made absolutely certain that the instillation is done properly so that the effect can be obtained. Certainly there is no objection to instilling the second drop, either of the cycloplegic or the paredrine, and almost routinely I have been using a second drop always of the paredrine solution.

Again I think that we ought to emphasize the need that was mentioned by Dr. Rutherford in accuracy in the subject of tests; that is, being absolutely certain the patient is cooperating and giving you or the examiner the proper answer, and is not in too much of a hurry to make a reply. I think that repeated questioning in this respect is absolutely essential in order to determine the accuracy of either the far point or the near point in making the examination.

Again with regard to dilatation of the punctum, I found that this was not a complication with the use of paredrine, and I don't think that with the use of paredrine that it makes any difference what drug is used before or after the use of homatropine or atropine.

As to children of school age, or below the age of, say, fifteen or sixteen years, I found that it is perfectly satisfactory to use the one drop of one per cent solution of atropine sulphate, followed in three or four minutes by a drop or two of one per cent aqueous solution of the paredrine hydrobromide. The results obtained are practically the same as those that are obtained after the repeated instillation of atropine sulphate. As a matter of fact, I believe that we really make the numerous or repeated instillations in the ordinary way first of all because they are made at home, they are instilled by a parent who doesn't know any too much about the proper method of instillation even though it has been explained to her, and it is done principally to insure at least a couple of drops being instilled properly.

Lastly, certainly if we have a method that gives us in office practice a procedure that can obtain the same results with a rapid recovery, such as has been shown to take place in these cases, I agree that we have something that is somewhat of an advance in our method of producing cycloplegia in refraction.

DR. LYLE S. POWELL, in closing: Mr. Chairman, I wish to thank the discussors. They have been very kind. I have only a few things I wish to say. The matter of paredrine has been injected into the discussion and I wish only to mention in passing that we have conducted parallel studies with paredrine. In calibrating these drugs with patients, however, we found that there was a slight but definite decrease in accommodation when used for mydriasis only.

We felt that inasmuch as benzedrine and homatropin in combination were entirely competent in the production of practical cycloplegia, we would not report on paredrine at this time. We do think, however, that it is a valuable drug but it is not in our opinion as valuable for use in my-

driasis only, because we found a slight reduction in accommodation which we did not find with benzedrine.

I agree with a great many of the things that have been said about atropine, and I am forced to agree with the ones who use one drop. I think that is part of the art of the practice of medicine — one develops his own technic and the way he can use things the best, and to me that is one of the non-essentials. The fact remains that to us, at least, this benzedrine and homatropin combination has resulted in a good, practical method of cycloplegia.

THE MECHANISM OF OPTOKINETIC NYSTAGMUS*

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THE FACT that optokinetic nystagmus is relatively little used as a method of clinical examination, seems to be due mainly to the insufficient knowledge of the normal mechanism of this phenomenon. One finds rather contradictory statements in the literature. On the one hand it is emphasized by most clinicians following Bárány[1] that the concentration of the subject's attention upon moving objects is a prerequisite for the production of optokinetic nystagmus so that it is considered a reaction taking place in the cerebral cortex; on the other hand it is regarded by Bartels[2] as a subcortical reflex, and there exist also observations of optokinetic nystagmus in the newborn, in idiots and in unconscious patients (Bartels[2]; Borries, Nordman and Liéou[3]).

This contradiction found an explanation, at least to a certain degree, by experiments of Ter Braak[4] indicating that two types of optokinetic nystagmus must be distinguished: the passive-nystagmus ("Stiernystagmus") and the active-nystagmus ("Schaunystagmus").

The passive-nystagmus is independent of attention and is reflexly provoked by the movement of retinal images; it may be observed not only in man but also in experimental animals (monkeys, dogs, cats, rabbits). In order to produce this nystagmus it is necessary that all or at least the majority of the objects within the visual

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field move in the same direction, since non-moving objects in the visual field prevent the appearance of this nystagmus, their retinal images moving in the opposite direction and forming an opposite stimulus if the eyes follow the moving objects. This passive-nystagmus may still be elicited after extirpation of the cerebral hemispheres in rabbits, dogs, monkeys; thus it is a subcortical reflex, and one may designate it *"subcortical type of optokinetic nystagmus."*

The active-nystagmus is produced only if objects move in the visual field which attract the attention, even if these objects form only a part of the objects of the visual field. This nystagmus is found in man, in monkeys and dogs, but not in rabbits. It is abolished in dogs and monkeys by bilateral extirpation of the cortical centers of the retinae in the occipital lobes; thus it is a cortical reflex and we may designate it *"cortical type of optokinetic nystagmus."*

One has, therefore, to distinguish between two types of optokinetic nystagmus, a subcortical and a cortical one. As to the mechanism of the latter, the centrifugal arm of its reflex arc originates in the occipital lobe (Cords[5]); it takes its further way over the vestibular nuclei, as far as the innervation of horizontal optokinetic nystagmus is concerned, since experiments of Spiegel[6] showed that horizontal ocular movements produced by stimulation of the occipital lobes depend on the intactness of the vestibular nuclei.

The question presents itself whether the subcortical type of optokinetic nystagmus uses a similar mechanism. In order to answer this question the following experiments were undertaken.

METHOD

The experiments were performed on cats and dogs. For observation of the optokinetic nystagmus the animals were examined in a dark room, with the head fixed. A paper cylinder (diameter 1.50 m., height 1.60 m.) with alternating vertical black and white stripes of 10 cm. width on its inner surface, was slowly rotated around the animal (usually one rotation in ½ minute) by an assistant standing outside the cylinder, while the experimenter with the animal was inside of the cylinder.

On the upper edge of the cylinder several strings were fixed converging toward a cord which was conducted over a pulley in the ceiling. This cord represented the axis of rotation, and the

cylinder was raised and lowered by it. The lamp illuminating the inner surface of the cylinder was placed behind the animal; the observer stood on the side of the animal board but somewhat behind the animal's head so that he might observe the movements of the corresponding eye without interfering with the animal's visual field which was filled by the moving stripes of the cylinder.

OPERATIVE PROCEDURES

In order to reach the roof of the midbrain the occipital lobe was exposed on the respective side and gradually pushed lateralward by inserting pledgets of cotton between the medial surface of the occipital lobe and the falx cerebri and the tentorium cerebelli respectively. The anterior part of the tentorium usually had to be removed to make the whole superior colliculus visible, which was then destroyed by electrocautery.

The floor of the fourth ventricle was exposed in the usual way by opening the posterior atlanto-occipital membrane, removing the adjacent part of the occipital bone and gradually elevating the vermis cerebelli from the floor of the ventricle. The lesion of the vestibular nuclei was produced either by incising with a small bent knife on the inner side of the restiform body, or by placing a heated wire alongside the medial aspect of this structure.

RESULTS AND COMMENT

First it may be stated in agreement with Ter Braak's[4] experiments that injuries to the occipital lobes do not prevent the appearance of the passive-nystagmus. Since the exposure of the superior colliculi necessitated the pushing lateralward of the occipital lobes, it was particularly ascertained in control experiments whether this procedure in itself impairs the passive-nystagmus. Exposure of the roof of the midbrain by pushing one occipital lobe lateralward often diminishes the frequency of the optokinetic nystagmus to both sides (compared at the same speed of rotation of the turning cylinder before and after the operation). This is particularly observed if the nystagmus is tested soon after the operation and may be due partly to the effect of anesthesia, partly to the effect of "shock" upon the lower reflex centers or to the loss of facilitating corticofugal impulses. But there appeared at most only slight differences between the optokinetic nystagmus to the left and that to the right side. In the experiment described below (Cat IV) for instance, fifteen minutes after exposure of the midbrain from the

left side the frequency of the optokinetic nystagmus to the right, produced by counterclockwise rotation of the cylinder at a rate of one turn per 30 seconds, was 5 per 30 seconds; the speed of the optokinetic nystagmus to the left (produced by clockwise rotation of the cylinder at the same rate) was 7 per 30 seconds. This could suggest that lesions of the left occipital lobe impair the optokinetic passive-nystagmus to the right side similarly as found by Rademaker and de Kleijn[7] for the active-nystagmus. Twenty-five minutes later, however, the opposite was found, a higher frequency of the nystagmus to the right (13 per 30 seconds) than of the nystagmus to the left (11 per 30 seconds). Thus such slight lesions of one occipital lobe as may result from exposure of the roof of the midbrain do not produce definite permanent differences between the passive-nystagmus to the left and that to the right side.

Unilateral lesion of the colliculus superior diminished the passive-nystagmus to the opposite side (i.e., with the quick component to the opposite side), while after bilateral lesions of this ganglion, at most only rudimentary reactions could be elicited by the optokinetic impulses. This is illustrated by the following experiments:

CAT IV

		Rotation of Cylinder (1 turn in 30 seconds)	
		(Clockwise) →	(Counterclockwise) ←
Time		*Nystagmus (Frequency per 30 Seconds)*	
11/12/37			
Before Operation		33 to left	33 to right
10:55 A.M.	Left occipital lobe exposed (ether anesthesia), pushed aside, left corpora quadrigemina exposed. Occipital lobe replaced in original position.		
11:05		7 to left	5 to right
11:40		11 to left	13 to right
12:00	Left colliculus superior cauterized.		
3:15 P.M.	Cat walks; light reflex: Right pupil contracts from 3 to 1 mm. Left pupil contracts from 3 to 2 mm. (tonic contraction particularly on left side).		
3:20-4:00		10-11 to left 9 to left 16 to left 17 to left 19½ to left	4 to right 1 to right 12 to right 13-14 to right (Animal is excited) 12½ to right
11/13/37	*Spontaneous Nystagmus* To left (13-17 jerks per 30 seconds)	19 to left 21 to left 28 to left	14 to *left* 7 to left 10 to left

SUMMARY CAT IV

Exposure of left superior colliculus does not produce definite differences between passive-nystagmus to left and that to right. Cauterization of left superior colliculus impairs the passive-nystagmus to right; then spontaneous nystagmus to the left develops. The optokinetic stimulus, produced by clockwise rotation of the cylinder increases this spontaneous nystagmus to the left. The counterclockwise rotation of the cylinder decreases somewhat this nystagmus but fails to produce nystagmus to the right.

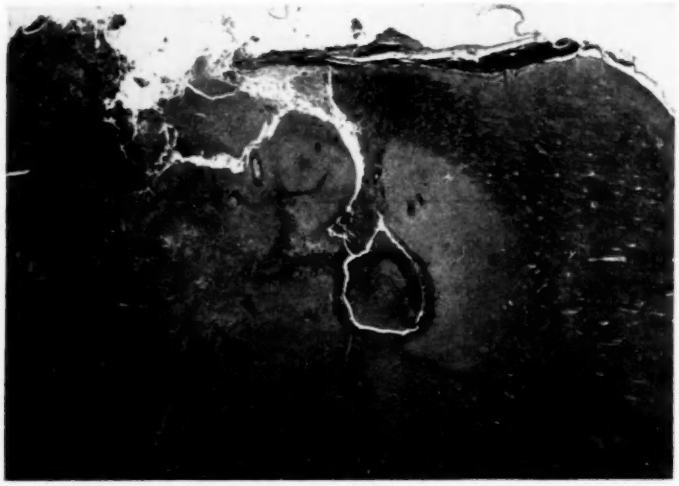

FIG. 1 — (Scala and Spiegel) — Cat IV — Destruction of left tectum (section in the region of transition between inferior and superior colliculi).

Anatomic Examination: (Fig. 1.) The left occipital lobe is slightly prolapsed; left superior colliculus and cranial part of inferior colliculus are cauterized. On cross sections the tectum of the left inferior colliculus is destroyed in its anterior part, and the tectum of the superior colliculus in its caudal part. In the anterior part of the left superior colliculus only the superficial layers are affected.

CAT XIII

		Rotation of Cylinder (1 turn in 30 seconds)	
		(Clockwise) →	(Counterclockwise) ←
Time		*Nystagmus (Frequency per 30 Seconds)*	
5/10/38			
Before Operation	Head in normal position.	30 to left	31 to right
	Head in side position. (Left ear up; right ear below).	37 down ↓	8 up ↑ (to upper lid)
3:00 P.M.	Cauterization of both corpora quadrigemina anteriora (Nembutal anesthesia, 36 mg. per kilo body weight).		
5/11/38 10:00 A.M.	Pupils 2 mm. diameter, dilate on excitation of the animal; in this stage tonic contraction by illumination.		
	Head in normal position.	No nystagmus	No nystagmus
10:10	Head in side position. (Right ear up.)	1 jerk up ↑	Eyes deviate to right canthus.
10:15	Head in side position. (Right ear up.)	No reaction	No reaction
10:20	Head in side position. (Right ear up.)	Slow deviation to right canthus.	Very slight deviation to right canthus.

Labyrinth Stimulation by Rotation on Bárány Chair

After 10 *Rotations to* Left: Eyes deviate to left, remain in this position for several seconds, then nystagmus to right with small amplitude develops.

After 10 *Rotations to Right:* Tonic deviation of the eyeballs to right, followed by nystagmus of small amplitude to left.

5/12/38	Pupils: 4 mm. diameter; light reflex sluggish, contraction to 3 mm. Spontaneously one eye jerk in 30 seconds to left.		
		Rotation of Cylinder (1 turn in 30 seconds)	
		(Clockwise) →	(Counterclockwise) ←
10:30	Head in normal position.	*Eye Reactions (per 30 Seconds)* 7 slow movements to left.	1 jerk to *left*
10:33	Head in normal position.	2 to left	Eye deviation to left
10:35	Head in normal position.	2 to left	Eye deviation to left
10:40	Head in normal position.	No spontaneous eye jerks.	No spontaneous eye jerks.
10:41	Head in normal position.	3 to left	No reaction
10:43	Head in normal position.	No reaction	
10:46	Head in normal position.	1 to left	1 weak jerk to left
10:50	Spontaneously, 1 eye jerk in 30 seconds toward left canthus.		
10:51	Head in side position (left ear up).	No change of spontaneous jerks to left canthus.	Spontaneous jerks to left canthus somewhat weaker.

SUMMARY CAT XIII

After cauterization of the superior colliculi the optokinetic stimuli produced (with the head in normal position) first no ocular movements, although the eye muscles reacted to labyrinthine stimuli. Later spontaneous jerks to left appeared, which were somewhat increased by clockwise rotation of the cylinder, and practically not influenced by counterclockwise rotation. In the side position of the head the optokinetic impulse only once produced a single upward jerk of the eyes (toward upper lid). Then the optokinetic stimuli produced, instead of upward or downward movements of the eyes, only slight deviation to the right canthus; later they failed to change the spontaneous jerks to the left.

Anatomic Examination: The region of the superior colliculi is covered by necrotic material. On transverse sections through the superior colliculi and the anterior parts of the inferior colliculi the lesion (areas of coagulation necrosis interspersed with hemorrhages) reaches the level of the aqueduct and third ventricle respectively. (Fig. 2.)

Thus the transmission of the optic impulses producing the passive-nystagmus to the effector apparatus depends on the intactness of the superior colliculi. Since it was shown in former experiments that the

FIG. 2.—(Scala and Spiegel).—Cat XIII.—Necrosis and hemorrhages in both superior colliculi.

vestibular nuclei play an important role in transmitting impulses for horizontal conjugate ocular movements from the frontal lobe (Spiegel and Teschler)[8] as well as from the occipital lobe (Spiegel)[6] to the eye muscle nuclei, it seemed of interest to ascertain whether the vestibular nuclei have a similar importance for the innervation of this subcortical nystagmus that depends on impulses transmitted through the superior colliculi.

Brunner[9] studying optokinetic nystagmus in patients with lesions of the central vestibular systems arrived at the conclusion that so-called inversion of the optokinetic nystagmus (nystagmus in the direction of rotation of the cylinder or failure of the optokinetic nystagmus to appear) is found only in patients with non-labyrinthine nystagmus. In patients with nystagmus due to lesion of the peripheral labyrinth or of its centers, "inversion" of the optokinetic nystagmus does not appear, according to Brunner.

Our experiments indicate that the situation is somewhat more complicated as far as the relationship of central labyrinthine nystagmus and subcortical optokinetic nystagmus is concerned. If one arranges the material, progressing from the slightest to the most pronounced disturbances the following types may be distinguished.

1. *Optokinetic Nystagmus Can Be Elicited in Both Directions*

   A. Optokinetic and vestibular nystagmus beat rather independently of each other. The jerks of the optokinetic nystagmus, opposed in direction to those of the spontaneous nystagmus, appear in the intervals between the periodically beating spontaneous nystagmus (illustrated by experiment II).

CAT II

		Rotation of Cylinder (1 turn in 30 seconds)	
		(Clockwise) →	(Counterclockwsie) ←
Time		*Nystagmus (Frequency per 30 Seconds)*	
11/8/37 Before Operation		*22* to left	*22½* to right
11:00 A.M.	Incision on inner side of both inferior peduncles (ether anesthesia)		
11:20	*Spontaneous Nystagmus* (Frequency per 30 seconds) 39 (rotatory and horizontal to the right)		
11:38		Spontaneous nystagmus not changed by optokinetic impulse.	48 horizontal to right.
11:45		13 rotatory to right and 6 horizontal to left.	
11:55		Only rotatory nystagmus to right.	
2:45 P.M.	11-17 rotatory nystagmus to right.	12 horizontal to left and 14 rotatory to right.	
2:55		18 horizontal to left; 11 rotatory to right. The jerks to left appear in the intervals between the spontaneous nystagmus to right.	20 horizontal to right and 12 rotatory to right.
11/9/37	5 per minute (mixed vertical and rotatory to right)	19-25 to left and 4 up ↑	19-27 to right and 4 up ↑

SUMMARY CAT II

After lesion of the vestibular nuclei, spontaneous rotatory nystagmus to right develops (first with horizontal component to right, later with vertical component). Optokinetic nystagmus can be produced in both directions, the jerks of the optokinetic nystagmus to left appearing in the intervals between the groups of spontaneous beats to right.

Anatomic Examination: (Fig. 3.) Extensive lesion in the caudal part of the right nucleus of the descending vestibular root; the lesion encroaches in the anterior parts of the oblongata upon the restiform body.

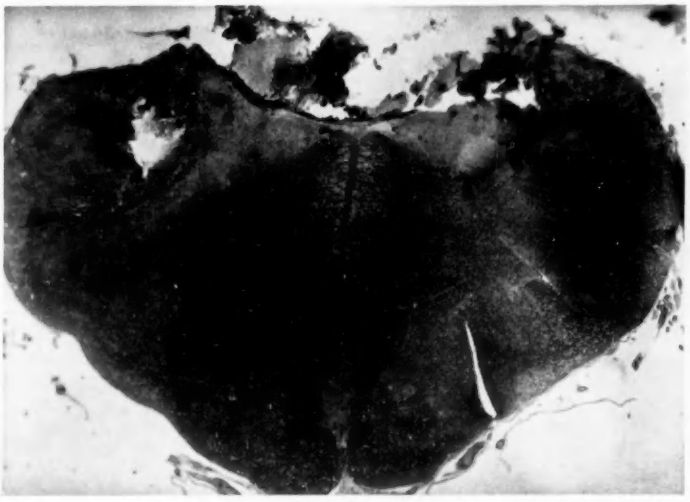

FIG. 3.—(Scala and Spiegel) — Cat II.—Right side (R): Right lesion in the nucl. of the descending spinal vestibular root. Left side (L): Left lesion extending from ventricle into nucl. of descending vestibular root.

On the left side there is only a small lesion extending from the ventricle into the nucleus of the descending vestibular root in its most caudal part.

Similar observations were made in Dog IX. Seven days after incision on the inner side of the inferior peduncles he showed spontaneous nystagmus to left, appearing in groups of 2-3 beats with a total frequency of 12-19 per 30 seconds. Clockwise rotation of the cylinder increased the frequency of the nystagmus to left to 25; on counterclockwise rotation there appeared between the groups of jerks to left also jerks to right (14 to left and 12 to right in 30 seconds).

Incomplete lesions in the cranial part of the vestibular nuclei inducing vertical nystagmus (Cat V) also do not prevent the production of horizontal optokinetic nystagmus to either side.

B. The optokinetic impulses influence the spontaneous nystagmus. They may increase and also inhibit it, as shown by the following experiment.

DOG X

		Rotation of Cylinder (1 turn in 30 seconds)	
		(Clockwise) →	(Counterclockwise) ←
Time		*Nystagmus (Frequency per 30 Seconds)*	
5/5/38 Before Operation		23-31 to left	26-27 to right.
11:00 A.M.	Bilateral incision on inner side of restiform bodies.		
5/9/38	*Spontaneous Nystagmus* (Frequency in 30 seconds)		
10:00	3-7-14 jerks to left alternating with 5-9 jerks to right.		
10:01		16 to left; 0 to right.	15 to right; 3 to left.
10:10	4 to right; 3-4 to left.		
10:11		8 to left; 5 to right.	15 to right; 7 to left.
10:20	2 to left; 7 to right.		
10:21			8 to right; 2 to left; 7 to right; 3 to left.
10:30	4-5 to left; 2-4 to right.		
10:31		11 to left; 0 to right.	7 to right; 0 to left.

SUMMARY DOG X

After incision on the inner side of the restiform bodies nystagmus developed beating in alternating groups to left and to right side. The optokinetic impulses increased the jerks opposite the rotation of the cylinder, sometimes also inhibited the jerks in the direction of rotation.

Anatomic Examination: The left incision is divided in its caudal end in two parts, a medial reaching into the nucleus dorsalis X and a lateral between tract. solitarius and nucleus of the descending root of the eighth nerve. It remains through the oblongata medially from this nucleus, partly also encroaching upon it. The dorsal part of the reticulate substance

is affected by small hemorrhages. The right lesion in its caudal part reaches from the ventricle into the tract. solitarius and the medial border of the nucleus of the descending root of the eighth nerve. On more cranial sections it destroys this nucleus and also the nucleus Deiters.

One finds here a type of spontaneous nystagmus that perhaps may be called "alternating nystagmus": groups of beats to one side alternating with groups of beats to the opposite side. The optokinetic impulses may increase the frequency of the beats in the direction opposite to the direction of rotation of the cylinder. Sometimes they may also inhibit the beats in the direction of rotation.

Occasionally the optokinetic impulses may facilitate the appearance of jerks in abnormal direction (vertical in the beginning of experiment on Cat I in the next group) and eventually they may reverse the direction of the spontaneous nystagmus (Cat XI in side position of the head in the next group).

11. *Optokinetic nystagmus can not be elicited in one direction (Experiments I and III) or in either horizontal direction (Experiment XI).*

This can be observed not only with large injuries extending from the vestibular nuclei into the reticular substance (Cat I) but also with small lesions limited to the vestibular nuclei (Cat III).

CAT I

		Rotation of Cylinder (4 turns per minute) (Clockwise) →	(Counterclockwise) ←
Time	Bilateral incision on inner side of inferior peduncles. Artificial respiration for ½ hr. necessary.	*Nystagmus*	*Nystagmus*
10/28/37 3:00 P.M.			
3:30	*Spontaneous Nystagmus* Rotatory to left with down ↓ component (13 per minute) and 3 purely down ↓		
3:31		Rotatory nystagmus to left (15 per minute) and 3 up ↑	Rotatory nystagmus to left 21; 3 down ↓ 1 up ↑ (per minute).
4:00	Rotatory to left 15-18.		
4:01		20 to left	15 to left
4:45	Rotatory to left, with down ↓ component 13-15 (30 seconds).		
4:46		12 rotatory to left (30 seconds)	14 to left and 1 up ↑ (30 seconds)
4:49	23-34 (30 seconds)	12-13 (30 seconds)	29 (30 seconds)
4:55	15 to left and 1 down ↓		
4:56			19 to left
10/29/37 10:00 A.M.	Rotatory to left and down ↓ 6-7 (30 seconds).	*Rotation of Cylinder (3 turns in 30 seconds)*	
10:01		9-10 to left and up ↑	6 to left
10:05	Rotatory to left and down ↓ 2-3, and 1 purely up ↑ (30 seconds).		
10:06		*Rotation of Cylinder (2 turns in 30 seconds)* 8 to left (with slight rotatory component)	
10:10	7-8 rotatory to left; animal excited.		
10:11			17 to left, rotatory and down ↓
10:15	6-7 rotatory to left and down ↓		
10:16		11 to left, horizontal and rotatory with down ↓ component and 1 purely up ↑	
10:18	3-6 horizontal and rotatory to left and 1 purely up ↑ (30 seconds).		
10:19		5 to left	10 to left (horizontal and rotatory) and 1 up ↑ (30 seconds)
10:20			6 to left and 1 up ↑
10:23	6-8 horizontal and rotatory to left and 1 up ↑ (30 seconds)		
10:24		10 horizontal and rotatory to left.	6 horizontal and rotatory to left.

SUMMARY CAT I

The lesion of the vestibular nuclei produced rotatory nystagmus to left with down component and some purely down jerks, later also up jerks. *The optokinetic stimuli fail to produce nystagmus to right.* Often but not regularly they increase the frequency of the spontaneous nystagmus to left. This was observed on clockwise as well as on counterclockwise rotation of the cylinder. In some experiments a few up jerks appeared on rotation of the cylinder, while the preceding spontaneous nystagmus showed, besides the rotatory nystagmus, only down jerks.

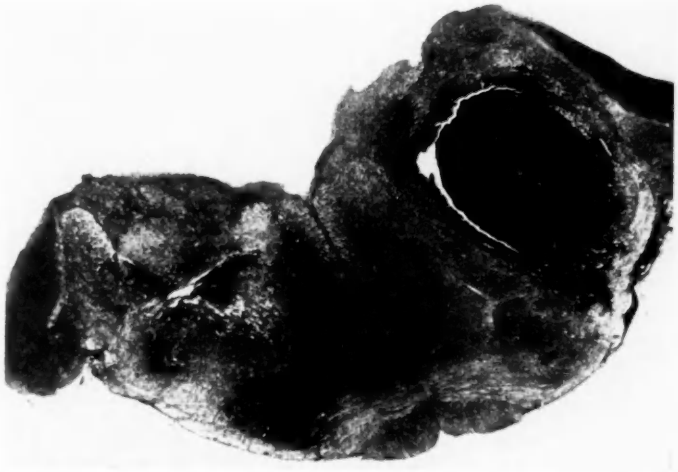

FIG. 4.—(Scala and Spiegel)—Cat I—On right side (R): Large hemorrhage in right reticulate substance. On left side (L): Lesion laterally and ventrally to left triangular nucleus.

Anatomic Examination: (Fig. 4.) The lesion on the right side starts in the right nucleus dorsalis vagi, forms a hemorrhage in the lateral part of the triangular nucleus and encroaches upon the dorsolateral part of the reticulate substance where it is found cranialward up to the level of the facial nucleus.

On the left side there is a puncture in the laterocaudal part of the triangular nucleus, extending on more cranial sections along the ventral border of this nucleus and into the adjacent most dorsal parts of the reticulate substance..

CAT III

		Rotation of Cylinder (1 turn in 30 seconds)	
		(Clockwise) →	(Counterclockwise) ←
Time 11/9/37 12:00	Cauterization of the vestibular nuclei by applying a heated needle alongside the inner border of the inferior peduncles.		
	Spontaneous Nystagmus (Frequency in 30 seconds)	*Nystagmus (Frequency in 30 Seconds)*	
3:00 P.M.	Horizontal and rotatory to left 23-26 and 1 up ↑		
3:01		28 horizontal and rotatory to left and 1 up ↑	22 horizontal clockwise to left and 2 up ↑
3:10	Horizontal rotatory to left 26 (one of these has vertical component)		
3:11		30 horizontal and rotatory to left (4 of these jerks have up ↑ component.	26 horizontal and rotatory to left (4 of these jerks have up ↑ component).
3:20	Horizontal and rotatory 26 to left (4 jerks have up ↑ component).		
3:21		31 horizontal and rotatory to left and 1 up ↑	21 horizontal and rotatory to left (4 with up ↑ component).

SUMMARY CAT III

The lesion of the vestibular nuclei produced horizontal and rotatory nystagmus to left; some jerks had also a vertical component. The optokinetic impulses fail to produce nystagmus to right. Clockwise rotation of the cylinder increases the nystagmus to left; counterclockwise rotation either fails to influence it or diminishes its frequency.

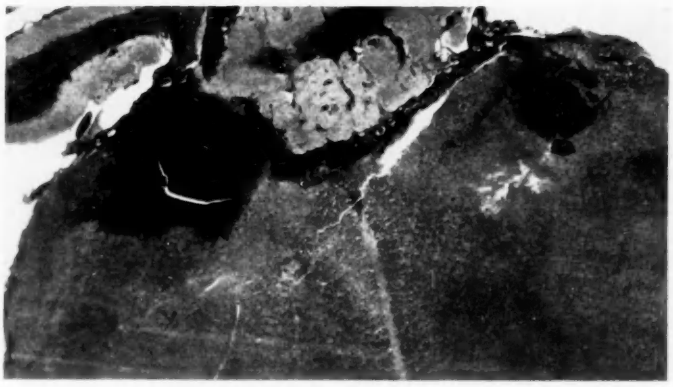

FIG. 5 — (Scala and Spiegel) — Cat III — Hemorrhage in either nuclei of the descending vestibular root, on the left side (L) more extensive than on right (R).

Anatomic Examination: (Fig. 5.) Bilateral lesion of the nuclei of the descending vestibular root in their caudal parts, more marked on the left side than on the right side. The lesions encroach upon the nuclei Burdach on either side, but leave the reticulate substance intact.

CAT XI

		Rotation of Cylinder (1 turn in 30 seconds)	
		(Clockwise) →	(Counterclockwise) ←
Time		*Nystagmus (Frequency in 30 Seconds)*	
5/7/38			
Before Operation	Head in normal position.	27 to left	29 to right
	Head in side position (left ear up).	25 down ↓	12 up ↑
11:30	Incision on inner side of cranial part of both inferior peduncles.		
5/10/38	*Spontaneous Nystagmus (Frequency in 30 Seconds)*		
10:15	Eyes downward deviated; 5 up ↑		
10:16		4 up ↑; increased amplitude.	
10:19	Eyes downward deviated; 2 up ↑		
10:20			6 up ↑
10:23	7 up ↑ (1 large, others small amplitude)		
10:24		6 up ↑ (small amplitude).	
10:26	7 up ↑ (Medium amplitude)		
10:27			9 up ↑ (2 large 7 medium and small amplitude).
10:30	4 up ↑ (1 large, 3 medium and small amplitude)		
10:31		8 up ↑	
10:34	6 up ↑		
10:35			11 up ↑
10:37	10 up ↑		
10:38		10 up ↑ and 1 to left.	
10:40	10 up ↑		
10:41			9 up ↑ and 1 to left.
10:45	8 up ↑		
10:46		9 up ↑ and 2 to left.	

HEAD IN NORMAL POSITION

Time			Rotation of Cylinder (1 turn in 30 seconds) (Clockwise) →	(Counterclockwise) ←
			Nystagmus (Frequency per 30 Seconds)	
10:48	HEAD IN NORMAL POSITION	8 up ↑		
10:49				8 up ↑
10:50			9 up ↑	
10:53		5–6 up ↑		
10:54			7 up ↑	
10:57		14 down ↓ (to lower lid and slightly to left canthus).		
10:58			First 2 down ↓; then 14 up ↑	
11:00		11 down ↓ and to left and 1 up ↑		
11:01				19 down ↓
11:04		9 down ↓		
11:05			1 down ↓ then 14 up ↑	
11:08	HEAD IN SIDE POSITION (RIGHT EAR UP)	9 up ↑; 8 up ↑		
11:10				10 up ↑
11:30		2 down ↓; 3 up ↑		
11:31			7 up ↑ (small amplitude).	
11:32				5 up ↑
11:34		3 up ↑		
11:35			8 up ↑	
11:37		6 weak and 3 distinct up ↑		
11:38				3 down ↑ (weak); then 8 up ↑ (distinct)

SUMMARY CAT XI

The lesion of the vestibular nuclei produced downward deviation of the eyeballs and upward nystagmus, which was transitorily changed by side position of the head into a downward nystagmus with a slight component to left. The optokinetic impulses failed to produce nystagmus to right and only occasionally produced single jerks to left (clockwise as well as counterclockwise rotation of the cylinder). They often increased the frequency and to a certain degree also the amplitude of the upward

nystagmus. In side position of the head the optokinetic impulse was able to increase as well as to reverse the spontaneous downward nystagmus, depending on the direction of rotation of the cylinder; the spontaneous upward nystagmus could be increased but not definitely reversed.

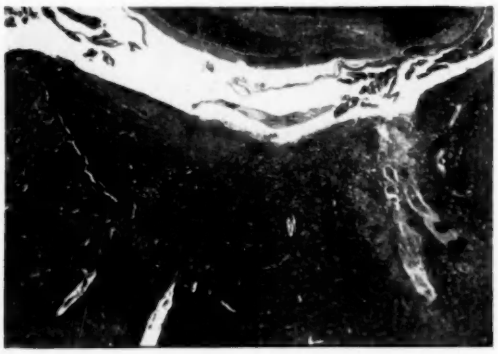

FIG. 6 — (Scala and Spiegel) — Cat XI — Incisions through the left triangular nucleus, left side (L), and in the lateral part of the right triangular nucleus, right side (R).

Anatomic Examination: (Fig. 6.) The left incision starts in the middle part of the oblongata where it lies in the triangular nucleus reaching into the dorsal part of the substantia reticularis; in more cranial sections it becomes larger and is found medially to Deiters' nucleus; it ends in the anterior part of the pons inward from the superior peduncle. The right lesion starts in the lateral part of the triangular nucleus, remaining medially to the nucleus of the descending root and Deiters' nucleus in the oblongata and beginning of the pons; then it lies more laterally and ends as a small hemorrhage dorsally to the nucleus motor.

CONCLUDING REMARKS

Reviewing these experiments, we find a variety of interference phenomena between the subcortical optokinetic nystagmus and the so-called spontaneous nystagmus that follows lesions of the vestibular nuclei or their fibers. The optokinetic impulses, if synergic in direction with the central labyrinthine impulses, may increase the spontaneous nystagmus; if antagonistic in direction, they may diminish and occasionally even reverse this nystagmus.

It is of interest to recall in this connection observations of Ohm[10] showing that the nystagmus produced by labyrinth stimulation through rotation is different in the light and in the dark. In case that the effect of fixation was excluded, Ohm's observa-

tions indicate that optic and labyrinthine impulses meet in some common center before reaching the motor nerves supplying the eye muscles; his observations do not allow, however, to determine more definitely the location of this interference of optic and vestibular impulses.* The hypothesis that this interference takes place in the vestibular nuclei is supported by the present series of experiments.

While small lesions of the vestibular nuclei do not interfere with the appearance of the subcortical optokinetic nystagmus, at least in the intervals between the groups of spontaneous jerks, larger lesions may prevent the production of optokinetic nystagmus in one or both directions, even if they do not extend into the reticulate substance.

The absence of optokinetic nystagmus in the direction opposite to a spontaneous central nystagmus may perhaps be explained by assuming that the optokinetic impulse is too weak to overcome the vestibular impulses. Such an explanation can not be applied if only vertical spontaneous nystagmus exists and horizontal optokinetic nystagmus can not be elicited.

The question may also be raised whether the absence of optokinetic nystagmus in these experiments may not be due to "shock" of some supranuclear center outside the vestibular nuclei (for instance in the reticulate substance) whose functional impairment may not be demonstrable by histologic examination. Such an explanation is rather improbable in view of the fact that, despite the absence of the optokinetic horizontal nystagmus except a few rudimentary jerks to the left in the above described experiments (Cat XI), the optokinetic impulses were still able to influence the vertical spontaneous nystagmus. Thus the lesion prevented the action of optokinetic impulses upon the motor apparatus of the eyes in regard to certain ocular movements only; it produced a circumscribed disturbance of function, not an impairment of the optomotor reflexes in toto as one should expect in the case of shock.

Thus these experiments suggest that the vestibular nuclei play an important role in transmitting to the eye muscle nuclei not only cortical voluntary impulses from the frontal lobes for conjugate horizontal ocular movements and impulses from the occipital lobes

* Ter Braak,[4] noticing similarities and interference phenomena between passive-nystagmus and labyrinthine nystagmus produced by rotation, also believes that the rhythm of both types of nystagmus probably is formed in a common center, but without determining its location.

innervating the optokinetic active-nystagmus, but also in transmitting the impulses from the superior colliculi for the subcortical optokinetic nystagmus (passive-nystagmus).

The anatomical details of the pathways by which the impulses descending from the superior colliculi reach the vestibular nuclei must still be elucidated. These impulses probably use tecto-bulbar and tecto-pontine bundles reaching the reticulate substance, and are thence transmitted to the vestibular nuclei. The connections of the vestibular nuclei with the segmental eye muscle nuclei represent the common path for labyrinthine ocular reflexes, voluntary ocular movements, and optokinetic cortical and subcortical nystagmus as far as movements in the horizontal plane are concerned.

The fact that the vestibular nuclei receive impulses from the superior colliculi seems of interest from another point of view also. Former experiments of Spiegel and Sato[11] showed that unilateral lesions of the vestibular nuclei produced in double-sided labyrinthectomized dogs quite similar phenomena as unilateral labyrinthectomy in normal animals (confirmed by Ferraro and Barrera[12] on macacus rhesus monkeys). Furthermore, it was shown by Spiegel and Démétriades[13] that Bechterew's compensatory nystagmus (appearing after extirpation of the second labyrinth following the first labyrinthectomy at two week intervals) is due to the development of an overexcitability of the vestibular nuclei on the side of the first labyrinthectomy. These phenomena indicate that the vestibular nuclei receive impulses not only from the labyrinth but also from higher parts of the central nervous system. One source of such impulse is, according to former experiments (Spiegel and Teschler[8]), the cerebral cortex; but also in subcortical ganglia such as the superior colliculi, impulses acting upon the vestibular nuclei may originate, as indicated by the present series of experiments.

SUMMARY

The mechanism of the subcortical type of optokinetic nystagmus (so-called passive-nystagmus) was studied in cats and dogs. The production of this nystagmus depends on the intactness of the superior colliculi, one-sided lesions of this structure impairing the nystagmus to the opposite side, while after bilateral lesions of this ganglion that the most rudimentary reactions could be elicited by the optokinetic impulses. After lesions of the vestibular nuclei various interference phenomena between the "spontaneous" cen-

tral and the optokinetic nystagmus were observed. The optokinetic impulses may increase the frequency of a spontaneous central nystagmus, if synergistic in direction, they may diminish its frequency or even reverse its direction, if antagonistic. Sometimes the jerks of the optokinetic nystagmus may be observed in the intervals between the periodically beating spontaneous nystagmus. Large lesions of the vestibular nuclei may prevent the appearance of horizontal passive-nystagmus in one or in either direction, even if the reticulate substance is spared. These experiments suggest that the vestibular nuclei play an important part in the mechanism of passive-nystagmus.

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DISCUSSION

Dr. A. Bielschowsky (by invitation), Hanover, N. H.: In the discussion of their excellent experimental work Dr. Scala and Dr. Spiegel refer to the contradictory statements in the literature concerning the question whether optokinetic nystagmus can be produced only if the subject's attention is concentrated upon moving objects so that optokinetic nystagmus would have to be considered a cortical reaction to the retinal stimuli, or whether it may be regarded as a subcortical reflex, a belief based on observations of optokinetic nystagmus in the newborn, in idiots and in unconscious patients; furthermore, on experiments showing that passive optokinetic nystagmus may be elicited after extirpation of the

cerebral hemispheres in rabbits, dogs and monkeys. Optic fibers conveying retinal stimulation from the primary optic centers (external geniculate body) to the corpora quadrigemina and the oculomotor nuclei have been supposed to represent the pathway for reflex movements of the eyes elicited by retinal stimuli. Whether or not this pathway is made use of not only in animals but also in man has been disputed for a long time. Since light perception in man is a function confined to the cortex, it is hardly conceivable that a movement of the eyes may be elicited by a retinal stimulus as a subcortical reflex, namely, without the participation of the occipital cortex in the process. The supposed existence of such a reflex mechanism would involve the assumption, that persons with cortical amaurosis could be induced to move their eyes at a certain angle, as well as in a certain direction, by having light in a dark room thrown onto a certain spot of their retinae. The movement brought about by the reflex mechanism under discussion would bring the peripheral image of the light to the fovea without the person perceiving the light. Not only is this inconceivable, but it never does occur in reality. In 1911 I had under my observation for several weeks and until she died, a patient with complete cortical amaurosis. The autopsy showed a circumscribed destruction of both occipital poles due to the obstruction of the two posterior cerebral arteries. During the observation her mental condition was absolutely normal. Despite the complete blindness the pupillary reaction to light was perfectly normal, showing the integrity of the reflex pathways connecting the external geniculate body with the oculomotor nuclei. Although the patient was able to execute to the normal extent both the voluntary and the commanded eye movements in any direction, moreover to direct her visual lines to her own hand or to an object she was touching, no eye movement from even the strongest retinal stimuli resulted, although she was warned to watch for a light flashing somewhere before her eyes. Such a behavior in a case of cortical blindness where the post-mortem confirmed the clinical diagnosis based on the integrity of the pupillary reaction to light and the absence of an optic atrophy, is an irrefutable argument against the assumption, that in man the retinal stimuli conveyed on subcortical pathways, that is without passing through the cortex to the oculomotor nuclei, may give rise to true reflex movements of the eyes. It is of course different in animals in which visual sensations originate not only in the hemispheres but in the optic lobes, so that ocular movements may be elicited by retinal stimuli after the extirpation of the hemispheres.

In 1899 Wilbrand's so-called prism phenomenon was recommended as a test for determining the site of the lesion in cases of hemianopsia. According to Wilbrand a patient with a lesion situated below the primary optical centers, that is, between the chiasm and the external geniculate body, would not respond with an eye movement compensating for a prismatic displacement of the retinal images toward the blind half of the retinae, while patients with hemianopsia due to a supranuclear, for instance a cortical lesion, were reported to respond to the same test with eye movements compensating for the prismatic displacement. In the latter group the positive result of the test was attributed to the integrity of the subcortical pathway connecting the primary optic centers with the motor nuclei. The fallacy of Wilbrand's prism test could be easily demonstrated.

According to Wilbrand patients with basal affections of the optic tract should not show a positive prism phenomenon because of the injury of optic fibers transmitting the stimulations of the corresponding halves of the retinae to the geniculate body. But in some unequivocal cases of tractus hemianopsia a positive result of the prism test could be obtained: the eyes moved in such way as to bring back to the fovea the image of the fixated light when it was displaced toward the blind periphery by a prism. Upon questioning, the patients stated that the light did indeed disappear when the prisms were put before the eyes, but that the apparent movement of the entire visual field resulting from the prismatic displacement of the retinal images indicated where the light was to be looked for and found again. On the other hand, when the prism test was used on unintelligent or inattentive persons it failed to bring about a compensative eye movement notwithstanding that their hemianopsia was caused by a supranuclear lesion which left the pathway between the geniculate body and the motor nuclei intact. For the time being I know of no irrefutable proof of the assumption that in man the above pathway must serve purposes other than for the light reaction of the pupils. Considering that retinal stimulation does not lead to eye movements unless the retinal image attracts the subject's attention, I cannot believe that any irrefutable proofs could uphold the production of the subcortical type of optokinetic nystagmus in man, because it is practically impossible to prevent the attraction of the patient's attention by the test.

The same considerations hold good for Cord's statement that lesions of the optic pathways and centers, the calcarine cortex included, have no influence whatever on optokinetic nystagmus provided that the centrifugal pathways descending alongside the centripetal fibers in the radiatio occipitothalamica to the motor nuclei of the eyes are intact. This statement of Cord's was based on cases in which optokinetic nystagmus could be produced despite a hemianopsia caused by lesions of either the optic pathways or their cortical terminations. If the optokinetic nystagmus was found to be reduced or could not be produced at all, either the cortifugal oculomotor pathways or their cortical origin were supposed to be involved, a supposition confirmed by autopsy in a few of the cases. But these few cases do not suffice for drawing definite conclusions as to the site of the lesion in hemianopsias from the result of optokinetic nystagmus tests. In my opinion an undisturbed optokinetic nystagmus would be just as incompatible with a complete hemianopsia as a positive reaction of the ocular muscles to retinal stimulation in cases of any complete amaurosis. But my argumentation is not be taken as an underestimation of the clinical value of optokinetic nystagmus. In cases of supranuclear disturbances of the ocular movements the investigation of optokinetic nystagmus is a necessary completion of the tests aiming at the site of the lesion.

The experimental work of Drs. Scala and Spiegel is likely to fill some gaps in our knowledge concerning the course taken by the innervations for optokinetic nystagmus. Whether or not they pass through the superior colliculi has been a controversial subject for many years. Bernheimer did not find any disturbance of the oculomotor movements in monkeys which he was able to observe for several weeks after the left colliculi had been extirpated. Referring to Bernheimer's statement, in 1926 Cord and other

authors denied that the superior colliculi play any part in the motor innervation of the eyes. The contrary opinion advocated by Probst, Beevor and Horsley, Minkowski and other authors, is supported by the experiments of Drs. Scala and Spiegel, which showed that the production of optokinetic nystagmus depends — at least in the experimental animals — on the integrity of the superior colliculi. Another portion of their work dealt with the part played by the vestibular nuclei in optokinetic nystagmus. Previous experiments undertaken by Spiegel and his collaborators had shown that the vestibular nuclei receive impulses not only from the labyrinth but also from the cortical centers governing the voluntary movements. Drs. Scala and Spiegel observed interference phenomena between the spontaneous central and experimental optokinetic nystagmus when the vestibular nuclei were injured, whereas greater lesions prevented the appearance of the subcortical type of horizontal optokinetic nystagmus. Whether or not the vestibular nuclei in man play the same part in the mechanism of the eye movements still remains to be seen.

The work of Drs. Scala and Spiegel gives many valuable suggestions for further studies of experimental nystagmus which may advance our knowledge of the mechanism of the ocular movements and contribute in facilitating the topic diagnosis of pathological cases.

Dr. W. B. Lancaster, Boston: I appreciate the compliment of being invited to discuss this subject. Like most of the rest of you, my knowledge of it is very, very meager.

I believe that nystagmus deserves more attention than we give to it. I look upon it as a defect of fixation. This is well illustrated by those cases of nystagmus in children with defective macula, therefore being unable to fix perfectly. Hence, the study of it will throw light on the physiology of fixation, a very important subject.

We must not think of nystagmus as a simple thing due to only one mechanism. Is it not probable that fixation involves many factors acting through many pathways between different parts of the central nervous system, what we used to call centers? All these areas and many others are embraced in what I am fond of calling the neuromuscular mechanism or ocular movements. In simplest terms, fixation occurs in this fashion: An image of some object falls on the periphery of the retina. The image attracts our attention and a reflex mechanism provides a movement so that the image falls on the fovea. If the movement, as is usual, is not exact, the image falls near the fovea, beyond it, still peripheral. By peripheral I mean any part of the retina outside of the macula.

Having gone beyond, the eye must be adjusted to move to bring it back onto the fovea.

Now this simple conception was my idea of the mechanism of nystagmus. In the case, for instance, of observing a series of moving objects passing by, as it were in procession from a car window, the eye would fix on one object, follow it along, and then be arrested by another which it would jump back to and follow that along, and this would be repeated indefinitely, as for instance in the set-up that the reader described of a

large paper cylinder in which the head is placed and observes these stripes moving steadily in front of the eyes.

As I say, that seemed to me a rather simple matter. I think our first explanations of things are apt to be too simple. One of the things that should strike our attention—but I confess I didn't think of it until I read this paper—is the regular rhythm with which we do that. Now perhaps the connection of the vestibular apparatus accounts for this regularity, this comparativeiy uniform rhythm with which we move the eyes back and forth. The vestibular apparatus is an old, we may say primitive, part of the nervous system. As new reflexes and new activities develop, this mechanism is utilized. Pathways from the central areas, that is from the frontal area for voluntary movements, from the occipital area for the visual motor reflexes, go through or are connected intimately with the vestibular area, and the performance is affected by this mode of action. In order for the impulses from the occipital visual motor cortical area, for instance, to reach the centers of the third, fourth and sixth nerves, the impulses follow pathways which these authors have studied. They have shown that the vestibular nuclei receive impulses not only from the labyrinth, as originally in the lower animals, but also from higher parts of the central nervous system. One course is the cerebral cortex; another is the subcortical ganglia, such as the superior colliculi. Thus the connections of the vestibular nuclei with the eye muscle nuclei represent the common form (a) labyrinthian ocular reflexes; (b) voluntary ocular reflexes; (c) optokinetic cortical nystagmus; and (d) optokinetic subcortical nystagmus, as least as far as horizontal movement and horizontal plane are concerned.

Thus, these experiments suggest that the vestibular nuclei play an important role in transmitting to the eye muscles and eye muscle nuclei not only cortical voluntary impulses from the frontal lobes for conjugate horizontal ocular movements and impulses from the occipital area for optokinetic active nystagmus, but also in transmitting the impulses from the superior colliculi for the subcortical nystagmus.

This has body not only for the ophthalmologist but for the neurologist, the neuro-surgeon, in helping him toward more accurate cerebral localization. My reaction to it is that it makes more evident than ever today the intricate interaction of the constituent parts of the neuromuscular mechanism for binocular vision, and the folly of those who still seek to explain all motor defects as a weakness of some one muscle.

SNAKE VENOM IN OPHTHALMOLOGY

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AMONG the 2,300 more or less known species of serpents, approximately 390 are called "poisonous" in the literal sense of the word ("serpentes venenosi" or "thanatophidia"), which may be considered as actively poisonous, and as such, dangerous to men and large animals. But besides these, notoriously poisonous, there are others also possessing a venenifluous system, but which are not included in the same group because the poison fangs are located in such a position that only by the merest accident can they injure men or animals.

Poisonous snakes belong to three distinct groups:

a) the *Opistoglyphae*, characterized by enlarged posterior maxillary teeth, having a longitudinal groove beginning near the excretive canal of the poison gland. These snakes are not generally classified as poisonous (Serpentes suspecti).

b) the *Proteroglyphae*, or *Elapidae*, characterized by the anterior maxillary teeth, differentiated and having a partly closed canal communicating with the poison gland.

c) the *Solenoglyphae*, with movable fangs, articulated, having a completely closed canal, and communicating also with the poison gland.

There remains the *Aglyphae* group which, although having a poison gland, lacks special teeth and consequently is even more inoffensive than the *Opistoglyphae*.

The poison has the twofold function of paralyzing and killing the victim, and of beginning the digestion (we must not forget that the animal is swallowed whole), so it must be inoculated into the live animal, in order to spread through the entire organism.

The venenifluous system is composed of a gland whose excretive duct begins in the base of the grooved tooth. This gland is similar to the salivary gland of mammals and is situated externally, behind the socket, is pear-shaped, and enclosed in a fibrous capsule, in which is inserted the anterior temporal muscle, whose contraction causes the forcible ejection of the poisonous liquid through the small canal of the tooth. The teeth of the *Viperidae* (of the Solenoglyphae group), being erectile, rise during the attack, which allows them to penetrate deeply into the tissues of the victim. The attack is made by a thrust of the snake, which extends itself like a spring, recoiling immediately to its previous position.

Beside serpents which inoculate their venon by a bite, there are others that project it to a distance, by the sudden contraction of the masseter muscle which compresses the poison gland.

The amount of poison that can be inoculated, its toxic activity, and the greater or lesser rapidity of its absorption, determine its degree of danger for men and large animals. Thus the most dangerous snakes are found among the *Solenoglyphae*, in the *Colubridae* and *Viperidae* families.

Snake venom is a mixed saliva composed of the secretion of the serous poison glands, together with that of certain supralabial mucous glands. It may be obtained separate from the product of the mucous glands by various methods, for instance, by letting the snake bite an impermeable membrane stretched over glass, or by pressing the edge of a Pettri-plate against the root of the teeth, etc. It is light colored, varying from milky to gray or yellow, according to the species of snake; it is viscous, and contains cellular impurities that sometimes make it turbid. The proportion of solid matter varies according to the species, being about 20 or 30 per cent. When dry, the venom takes a pseudo-crystallin form. In the dry state it keeps along time without deteriorating. It is, however, rapidly and intensely affected by ultra-violet rays, and to a lesser extent by the Roentgen and infra-red rays.

Besides water the venoms contain variable quantities of pigment, small quantities of substances soluble in ether, chlorides, phosphates and other mineral salts in smaller proportion, proteines coagulable by heat, proteoses and sometime peptones. Certain enzyms are the cause of some of its activities, and it is not yet known if its toxic power is due to small quantities of some highly destructive substance absorbed by the elements already known, or if it is produced by the very proteines already identified.

The way the different poisons act varies, according to the predominant group of elements, but in general the venoms contain the same active principles, only the respective proportion varying, and that to a notable degree.

According to Houssay the venoms of the various serpents may be grouped as follows:

I. Toxic action:
   a) Curarizing action (*Naja-naja* type)
   b) Neurotoxic action (*Crotalus terrificus* type)
   c) Shock producing action (*Lachesis* and *Bothrops* types)
   d) Coagulating action (*Vipera russelli* and *Bothrops* types)

II. Phosphatidasic action:
   a) Hemolytic action
   b) Inhibitary action of coagulation
   c) Cytolytic action

III. Protease action:
   b) Local irritation action, swelling, hemorrhage
   b) Hemorrhagic, grangrenous action (*Lachesis* type)

   In Group III pancreatic hemorrhages and fatty necrosis by injecting the pancreatic duct are also included, as well as distal hemorrhages, besides the diminution of fibrinogen in the blood stream (*Crotalus adamenteus* and *Lachesis flavoridis*)

IV. Thrombinic action — coagulation of blood. In this type alterations of erythrocytic resistance, reduction of precipitation rate, shock, drop of arterial pressure, leucopenia and alterations of the blood albuminoid bodies, are also included.

Ever since the times of empiric medicine, back to Galeno's reference to the cure of a leper who had taken wine from a barrel in which a snake had drowned, snake venom has been used as a therapeutical agent. In oriental medicine, this venom is included in the pharmocopea and in homeopathy the venoms of *Crotalus* and *Lachesis-muta*, occupy a prominent place.

Similar to Galeno's case, during the XVIII century and beginning of the XIX, there are several other cases of lepers who claimed to have been cured after snake bites. Based on these assertions, Mariano José Machado submitted himself voluntarily to the bite of a *Crotalus-terrificus*. This experiment was made before

several physicians in Rio in 1840 and resulted in the death of the patient in 24 hours. An interesting detailed description of the effects of the venoms was made out, but no mention was made of ocular disturbances.

Vital Brasil, in Saõ Paulo, injected into various lepers nonlethal doses of the venom of the *Crotalus-terrificus*, without obtaining any improvement. In the United States of America, A. Self, and afterwards Spengler, were the first to recommend the use of rattlesnake venom in the treatment of epilepsy and asthma, but the favorable results are much disputed, and recently Finkelmann verified the inefficacy of the venom of *Agkistrodom piscivorus* in the treatment of epilepsy with the product derived from that venom and called "Moceasinmulford." Peck and Sabotka used the venom of *Agkistrodom piscivorus* in the treatment of hemorrhagic diseases, its use having been extended by MacFarlane and Barnett to the treatment of haemophilia. The isolation of the coagulating principle of the venom of *Bothrops jararaca* by von Klobusitzky opened up a wider field to its use in the treatment and prevention of hemorrhages.

Monaelesser and Toguet, as well as Laignel-Lavastine, Calmette and Koressios, developed the idea of using *Naja* venom in the treatment of cancer and of Algias whether cancerous or not, the method being still the object of discussion and study. *Naja* venom was also employed against high blood pressure but with varying results.

The use of these various venoms in the treatment of the diseases mentioned is based on the study of their respective physiological properties, but in some cases, e.g., epilepsy, algias, etc., the results of its accidental use led the investigators to make further research. Thus, if the use of certain venoms to increase the coagulability of the blood arose from the knowledge of their different properties, the use of others in the treatment of epilepsy, cancer, etc., came from empiric observations of improvement in persons accidentally bitten. Thus the observation of the sedative action of the poison of a spider *(Lycosa)* on the pains of a leper, and mentioned by Monaelesser, led Calmette to further systematic studies of the calming action of the *Naja* venom, whose paralyzing effect on the sensory nerves was already known.

The properties of various venoms have been studied in the recent decades, and they are used according to their physiological action for a corresponding pharmacodynamic effect. The venoms

in which the neurotoxic element predominates are naturally indicated for the treatment of algias from their soothing effect upon sensitiveness (*Naja-naja, Crotalus terrificus,* etc.). Likewise the venoms in which the coagulating principle predominates are logically the most appropriate for the therapeutics of hemorrhagic conditions, and the proteolytic factors would naturally be the ones of interest in the use of venom in the treatment of malignant tumors.

As was already stated, together with desirable elements, the venoms contain useless ones, and only the isolation of the former can give a greater therapeutic value to snake venom. Their isolation has been obtained by the fractioning of the various component factors. Thus, the coagulating element of the venom of *Bothrops jararaca* was isolated by von Klobusitazky and Koenig and there are on the market pharmaceutic products derived from fractioned venoms, and with well defined pharmacodynamic properties ("Hemocoagulase" and "Bothropase"). Slotter and Fraenkel-Conrat, also by fractioning, succeeded in isolating the active principle of the venom of *Crotalus terrificus,* and in obtaining "Crotoxina," with an action similar to that of ferments, and which, while it acts upon the lipoids of the nervous tissue, acts likewise upon the lecithine of the blood cells causing hemolysis, combining therefore two of the properties of the venom of the *Crotalus terrificus,* neurotoxic and hemolytic, but without the coagulating property.

To reduce the toxicity of the venoms, various methods have been employed which, while diminishing their toxic and irritant force, still retain the useful properties. Among them is the weakening of the venom by formol, as Ramon did with toxins, for which reason the venoms thus de-poisoned are called anavenoms. Likewise, Esveld, making a very weak solution of the venom of *Naja sputatrix,* and inactivating it, prepared a pharmaceutical product of definite properties, and known on the market as "Cobratoxin." Similarly Brecher prepared a solution used in the treatment of trachoma and called "Trachozid." Still others (Vital Brasil, etc.) have prepared very dilute solutions of venoms, without other modifications, and used them for therapeutic purposes. It is well to remark here that the action of the venoms varies very much according to the dose. For instance, that of *Bothrops jararaca,* as von Klobusitzky found, has an anti-coagulating effect in strong doses, but is intensely coagulating in weak ones.

Snake casualties are relatively frequent. In India, according to Chopra and Iswariah, there are from 20,000 to 25,000 cases of death from snake bite. In Australia, Fairley mentions 240 fatalities. In Europe such casualties are rarer, and in Prussia from 1920 to 1925 there were but 150 cases of people bitten by snakes *(Vipera berus)*, only one case being fatal. In the United States of America, according to Hutchinson, there were in 1929 about 500 cases, with a mortality of about 16 per cent in those not treated, and of 3.75 per cent in those treated with serum. In Brazil, of 8,000 cases mentioned by Kraus, in about 20 years, the mortality was from 25 to 30 per cent in the cases not treated, dropping to 2.7 per cent in those treated by serum.

Ocular disturbances, in the case of snake poisoning, differ in nature and vary in frequency, according to the species of the serpent. The Institute of Butantan, at Sao Paulo, Brazil, which has been dealing with ophidism since the beginning of the century, tried to ascertain the various consequences of snake poisoning, and for this purpose distributes, together with the ampullae of curative serum a questionnaire in which are to be registered the various phenomena observed. Among the questions is the following:

"Was there blindness?"

In the 5,780 questionnaires at hand, ranging from 1902 to 1937, we find the following distribution of casualties caused by the different species, the first column giving the total number, the second the number of ocular casualties, and the third, the percentage relation:

Crotalus Terrificus Terrificus	643	387	60.1%
Lachesis muta	13	7	53.7%
Bothrops cotiara	64	16	25.0%
Micrurus corallinus (Proteroglypha)	12	3	25.0%
Bothrops jararacussú	610	121	19.8%
Bothrops alternata	351	61	17.5%
Bothrops neuwiedii	203	25	12.3%
Bothrops jararaca	2707	277	10.2%
Bothrops atrox	91	4	4.4%
Unidentified snake	1084	183	16.9%

All the serpents above referred to, with the exception of *Micrurus Coralinus* are Solenoglyphae. From the above data, one can clearly see the frequency of ocular troubles after the bite of a rattlesnake *(Crotalus terrificus)* which is further confirmed by common observation.

The ocular lesions described in reply to the question: "Was there blindness?" are very varied, and, if the description of the

ocular phenomena observed are fairly exact in many cases, in others there is merely the mention of a "darkening of the sight." The ocular symptoms described for rattlesnake poisoning include "darkening," appearing immediately after, and sometimes 6 hours after the bite. The functional disturbance varies, being sometimes only a dimming of the sight, sometimes a complete loss. The duration of the phenomena is also variable, from only a few minutes to more than ten days, disappearing generally immediately after the application of the serum. There have also been cases in which the visual disturbance appears precisely after the application of the serum. There has also been mention of conjunctival hemorrhages, edema of the cornea, impossibility of opening the eyes, paralysis of the ocular movements, squint, lid ptosis, monolateral blindness, mydriasis, etc.

The *Lachesis muta* caused edema of the cornea and sub-conjunctival hemorrhages.

The *Bothrops cotiara* caused xanthopsia, among other phenomena. The *Micrurus corallinus* caused lid ptoses, edema of the cornea, "darkening of the sight."

The *Bothrops jararacussú* caused the loss of sight, which happened immediately after the bite, and sometimes two days after, the disturbance being of variable duration.

There were also cases of conjunctival hyperhemia without other ocular disturbance, a feeling of pressure in the eyelids, lid ptoses, lid edema and photophobia.

The *Bothrops alternate* caused temporary blindness, without other external phenomena. It is an interesting fact that monolateral blindness was seen in three cases in animals (dogs and a cow).

The *Bothrops neuwiedii* and the *Bothrops jararaca* cause the appearance of functional disturbances that vary from the "darkening of sight" to its loss, the latter being in some cases intermittent. The appearance of visual phenomena also varies, coming sometimes immediately after the bite, sometimes 14 hours after. The duration may be from a few minutes to many hours. In some cases the appearance of blindness coincided with the injection of the serum. Pupillar mydriase was noticed, and also ocular pain, hyperhemia and defective venous circulation.

We tried to learn, from ophthalmologists residing in Brazil, what they had observed in regard to ocular manifestations in pa-

tients bitten by venomous serpents, and for that purpose sent a questionnaire to the 250 ophthalmologists whose names are registered in the last edition of *Indicia Medicorum*. We also extended our inquiry to several colleagues in Uruguay and Argentina. From the replies received there seems to be few observations of the ocular manifestations of ophidism, which is confirmed by the scanty references to the subject in the literature of this specialty.

My colleagues reported the following:

Odilon Alves[1] observed a case of uveitis with ocular hypertension in a patient bitten by a *Bothrops jararacussú* four months' previously. The patient felt pains in his eyelids immediately after the bite, having used anti-bothropic serum.

Durval Prado[2] noticed a patch of chorioretinitis, finely pigmented, in a patient who had been bitten two years before by *Bothrops jararaca*, and remained blind till the application of the serum. Later her sight returned, and she consulted a physician for phenomena of accommodation asthenopy, unaware of the lesion in the back of the eye.

T. Falcao[3] mentions the case of a Japanese who became blind and deaf in consequence of a snake bite, but who didn't know the species and refused to submit to an examination.

O. Carleial[4] observed a case of optic atrophy in both eyes with complete blindness in a man bitten by a *Crotalus Terrificus* two months before. Besides blindness, attributed to the bite, the patient had atrophy in contractures of the bitten limb.

Prof. J. Britto[5] reports that in his observation of 80,000 patients, whether in a private or a hospital clinic, he met only two cases of ocular lesions that were attributed by the patients to snake bites. In the first case there was anophthalmus of the right side following an enucleation in consequence of the snake bite in the very eye ball and in the second case atrophy of the eye ball on one side and sub-dislocated cataractous lens on the other eye, the patient saying that the trouble in his eyes had begun after the bite.

S. Martins[6] found blindness, conjunctival hemorrhages and edema of the cornea in a patient bitten by a *Crotalus terrificus terrificus*. This patient died 5 days after the bite, not having been treated with serum.

Prof. Linneu Silva,[7] in more than 30,000 cases, noticed only one case of retinal hemorrhage in a patient who had been bitten a few days before by a poisonous snake, species unknown.

H. Góes Ferreira[8] reports the case of a patient suffering from optic atrophy in both eyes who said it began after a snake bite, the other malignant effect of which had been cured by empiric treatment.

Pereira Gomes,[9] among 60,000 private and 30,000 hospital patients, over a period of 26 years, saw only one case of ocular disturbances arising from snake bite. This was from a *Crotalus terrificus* and caused a discreet hemorrhage with edema that extended even to the corneal epithelium, and also pupillar mydriasis. He could not ascertain if there was paralysis of the extrinsic muscles because of the patient's state of prostration, and it was necessary to force open the eyelids in order to examine them. Ophthalmoscopy hindered by edema of the cornea revealed slight hemorrhages of the retina in the visible parts, it being impossible to ascertain the state of the discs. There was a great reduction of visual acuity, which lasted throughout the effect of the poison. Even after recovery the toxic amblyopia continued, the sight eventually returning without any atrophy of the discs, or other noteworthy alteration. The patient received injections of specific anti-crotalic serum and recovered.

M. E. Alvaro,[10] among his private and hospital patients, more than 35,000 in number, saw only one case of ocular disturbances from snake bite, and there was only a certain difficulty in opening and moving the eyes, which seemed to the author to be of pithiatic nature. The patient, who had been bitten 16 times by *Crotalus* reported that each time he suffered from great functional disturbances which made him blind. The first times he had been treated with serum but afterwards he was already immune.

In the fundus oculi nothing abnormal was noticed, eye and lid movements being normal, as well as the pupillary reflexes. Vital Brasil[11] reports that the venom of the *Bothrops jararacussú*, contrary to what happens with that of the *B. jararaca*, causes visual phenomena characterized by the dimming of the sight. He says, also, that venom of the Elapineae causes lacrimation and temporary blindness, and that the venom of the *Crotalus* always causes visual disturbances. In the clinical records published by Vital Brasil there are various references to visual disturbances after snake bite, among them some that have a more detailed description. But the reference, found in medical literature, to the effect of snake venom on the eyes is generally quite scanty.

Boullet,[12] Encongneres,[13] and Magalhaes[14] in 1875 mention cases of blindness after snake bites, but do not describe the lesions noticed. Knies[15] suggested the hypothesis, not based on facts observed, that the ocular lesions are due to central hemorrhages, or in the eyeball itself. Lesions of extrinsic muscles were described by Richards.[16] DeVere-Condon and Rennie[17] mention ptosis and protrusio bulbi, and Berenger-Feraud[18] describes conjunctival and corneal changes from pure venom that had reached the eye. Hilbert[19] calls attention to xanthopsia appearing after snake bite. More recently Blatt[20] describes cases of ophidism in which he observed pupillary mydriasis, paresia of accommodation, palpebral ptosis and paresia of the extrinsic muscles. Bakker[21] and Schunek[22] mention ocular lesions from the venom of *Naja Tripudians* sputatrix and *Sacpedon hoemarchates* that entered the eye. Zannetin[23] and Sarnelli[24] found ocular lesions caused by the venom of *Naja nigrocollis*.

These last species of serpents, when attacked, are accustomed to project the venom to a distance. The eyes thus hit show whitish conjunctival changes due to the proteolytic action of the venom. There are also formations of milky-colored membranes. The lesions are very painful, but the inflammatory phenomena pass after 5 or 6 days, without leaving any traces. When the cornea is hit, it is more serious, with danger of losing the eye. The treatment consists of washing the injured part with serum and the application of the antiophidic serum paraenteral, local or subconjunctival.

Peratoner[25] noticed in patients bitten by vipers, slow pupillary reflexes and ptosis which promptly disappeared on application of the serum. Cesario de Andrade[26] mentions the constant appearance of ptosis palpebral in the cases of poisoning by serpents of the *Colubridae* group, giving as characteristic of the ocular disturbances caused by the poisons of the *Viperidae* mydriasis and pupillary paralysis with edema of the cornea; he also noticed the frequent disturbances of visual acuity. In the case of a bite by *Bothrops jararacussú*, he also noticed xanthopsia. Experimenting with the eyes of animals, Kellaway[27] found that even non-lethal doses of the venom of *Notechis scutatus* caused blindness. Koressios,[28] injecting *Naja* venom in a dose of 1.50 milligrams under the conjunctiva of rabbits, noticed after 7 minutes lasting miosis with paralysis of the light reflex. He also noticed a rise in the blood pressure and a dilation of the arteries at the fundus. After an intramuscular injection of 1.50 milligrams of *Naja* venom, he

observed in man a miosis of an hour's duration, accompanied, in one case, by a fall of the ocular tonus. This diminution, however, was not observed in other cases. He also noticed a slight rise in the blood pressure in the retina, more conspicuous in pathological cases; also an incipient contraction, followed by the dilation of the retinal vessels.

Giannatoni,[29] using solutions of *Naja* venom, in doses of 1.50 milligrams in instillations in the conjunctiva in subconjunctival retrobulbar and intra-muscular injections, found no action of the venom in the last two ways, contrasted with its application by instillation and subconjunctival injection, which caused a pronounced miosis and fall of the ocular tonus which lasted several days. He also observed that a repetition of the injections caused an increase of the hypotensor action, which extended to the other eye. He also made subconjunctival injections after the instillation of cocaine atropine and pilocarpine, noticing the inhibitions of the mydriatic and anesthetic action of cocaine, an increase of the mydriatic action of atropine, an increase of intensity without an increase in duration of the action of pilocarpine. In painful glaucoma, retrobulbar injections seem to ease the pains, but the effect is not lasting. Lebus and Hubert,[30] using *Naja* venom, noticed a lowering of ocular tonus.

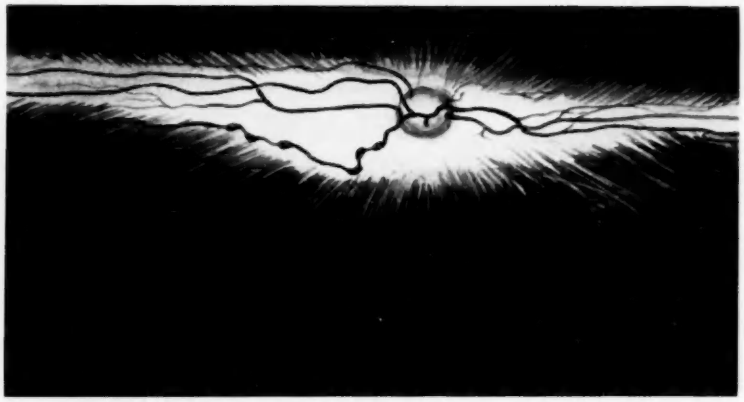

FIG. 1 — (Alvaro) — Fundus of Rabbit No. 684, 10 hours after paraenteral injection of sublethal dose of *Bothrops jararaca* venom. Ampuliform dilated artery can be seen.

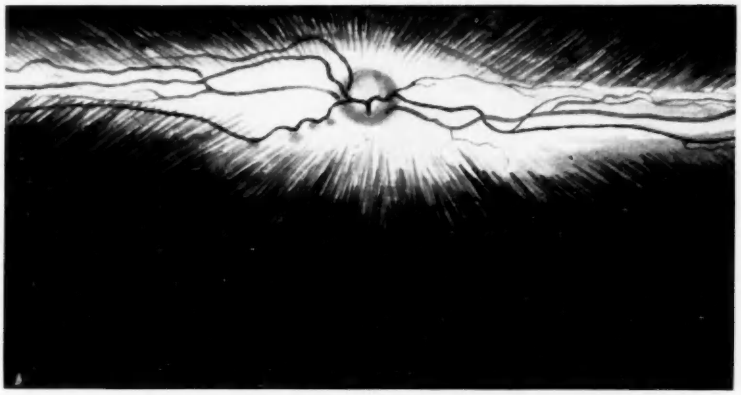

FIG. 2 — (Alvaro) — Fundus of Rabbit No. 684, 12 hours after paraenteral injection of sublethal dose of *Bothrops jararaca* venom. Minute hemorrhages can be seen.

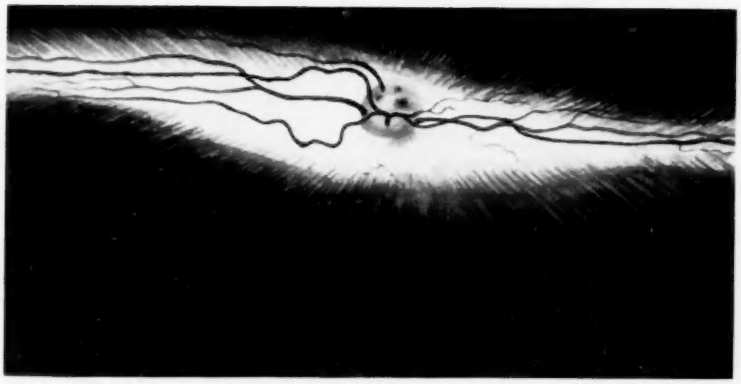

FIG. 3 — (Alvaro) — Fundus of Rabbit No. 682, 36 hours after paraenteral injection of sublethal dose of *Bothrops jararaca* venom. Small hemorrhages on the disc and a slight edema can be seen.

We made at the Pinheiros Institute the following experiments with rabbits *(Lepus-Orictolagus-Cuniculus)*:

1 — Rabbit No. 680. Subcutaneous injection of 1 cc. of a 2.0/00 solution of the venom of *Bothrops jararaca* corresponding to 2 milligrams of the pure venom. We noticed only a dilation of the conjunctival and retinal vessels. The ocular tonus remained unchanged. There was a slight initial miosis followed by retardation of the light reflex.

2 — Rabbit No. 681. Injection of 3 milligrams of the venom, 1½ cc. of the solution of *Bothrops jararaca*. There was no pupillar alteration, except the retardation of pupillary reflex after 12 hours, which lasted about 48 hours. Dilation of the conjunctival vessels. Retinal vessels normal. Ocular tonus unaltered.

3 — Rabbit No. 683. Injection of 4 milligrams of the venom of *B. jararaca*. After 12 hours here was a pronounced conjunctival hyperhermia and slight retinal hemorrhages. Slight ampulliform dilations in the vessels of the retina. Slow reaction of the pupil to the light. Slight subconjunctival hemorrhages.

4 — Rabbit No. 684. Injection of 5 milligrams of the venom of *B. jararaca*. After 10 hours there was a marked conjunctival and perikeratica hyperhermia. Retinal vessels dilated, with the formation of small ampullae and peripheral hemorrhages. Complete blindness developed 24 hours later, and edema of the optic nerve appeared after 36 hours. After 60 hours the edema disappeared and the retinal vessels were thinner. It seemed to see again. There was no alteration of the ocular tonus. Pupil dilated with weak and retarded light reaction. After 84 hours the pupillary reaction was normal and there were no more vascular alterations.

5 — Rabbit No. 682. Injection of 6 milligrams of the venom of *B. jararaca*. Eight hours later there was conjunctival and perikeratica hyperhemia ampulliform dilations of the vessels of the retina, and slight terminal hemorrhages. Vessels of the iris dilated. Pupillary reaction retarded and slow. Complete blindness 36 hours later with edema of the optic nerve and slight hemorrhages on the disc besides the other alterations. The choked disc disappeared after 60 hours, but the blindness persisted. The retinal vessels decreased in caliber and the perikeratic hyperhemia disappeared; 84 hours after there was still vascular dilatation, and blindness persisted. This rabbit died 10 days after the injection.

6 — Rabbit No. 686. Injection of 8 milligrams of the venom of *B. jararaca*. Six hours after there was choked disc vessels dilated, pupillary mydriasis, paralysis of the light reflex. Death of the rabbit 6½ hours after the injection.

7 — Rabbit No. 684. Injection of 5 milligrams of the venom of *B. jararacussú*. Ten hours after, the pupils showed a slow reaction, and there was a slight edema of the optic nerve. Retinal vessels dilated. Slight hemorrhages. Perikeratic hyperhemia, dilation of the visible

vessels of the iris. Sixty hours later there was still vascular dilatation and mydriasis. Eighty-four hours after, the hyperhemia had disappeared, as well as the choked disc; there were no signs of blindness.

8—Rabbit No. 198. Injection of 6 milligrams of the venom of *B. jararacussú*. Three hours later there was pupillary mydriasis, with slow reaction to the light. Ten hours after, we noted a slight choked disc and dilation of the retinal, conjunctival and iridian vessels. Absence of perikeratic hyperhemia. Sixty hours later there was still ampulliform dilatation of the vessels both of the conjunctiva and of the retina. The pupil was already in miosis with normal reflex. No signs of blindness.

9—Rabbit No. 319. Injection of 8 milligrams of the venom of *B. jararacussú*. There was mydriasis and slow reflexes. Ten hours afterwards there was dilatation of the retinal, conjunctival and iridian vessels and slight edema of the disc. Absence of perikeratic injection. Thirty-six hours afterwards there was still the ampulliform dilatation of the conjunctival and retinal vessels. Sixty hours afterwards, the vascular dilatation and the choked disc still persisted but the pupils were already normal. No signs of blindness.

10—Rabbit No. 828. Subcutaneous injection of 1 cc. of a 1.0/00 solution of the venom of *Crotalus t. terrificus* equivalent to 1 milligram of venom. Two hours afterwards we noticed that the light pupillary reflex was slower; in the fundus oculi nothing abnormal was noticed. Ten hours afterwards the retinal vessels were dilated, and there was a slight edema of the disc. The pupil was in miosis with normal light reflex. The rabbit died during the night.

11—Rabbit No. 394. Subcutaneous injection of 1½ cc. of a 1.0/00 solution of the venom of *Crotalus t. terrificus*, equivalent to 1.5 milligrams. Two hours afterwards the pupillary light reflex was slower in the fundus oculi. Nothing abnormal. Eight hours afterwards a slight pupillary moisis with normal light reflex. Slight edema of the disc. Retinal vessels contracted. Twenty-three hours afterwards there was a mydriatic pupil without light reflex. The retinal vessels were contracted with ampulliform dilatations and slight terminal hemorrhages. Slight edema of the disc. The visual function seemed to have ceased. There was paralysis of the hind parts of the animal. This rabbit died twenty-seven hours after the injection.

12—Rabbit No. 463. Subcutaneous injection of 2 cc. of a solution equivalent to 2 milligrams of the venom of *Crotalus terrificus*. Two hours afterwards the light pupillary reflex was slow. In the fundus oculi we noticed slightly dilated vessels with the blood clearer in color than normal. Six hours afterwards the vessels of the retina appeared very contracted with ampulliform dilatations. The pupil was very large with slow contraction movements in the light. The rabbit died 7 hours after the injection.

13—Rabbit No. 600. At 10:50 A.M. the ocular tonus was taken and showed 4 and 7, with weights of 5.5 and 7.5 in the *Schioetz* tono-

meter. At 11 A. M., injection of 2/10 cc. of a 20/00 solution of the venom of *B. jararaca* equivalent to 4/10 of a milligram, in the left eye, under the conjunctiva, after anesthesia, with a ½% solution of pantocaine. At 11:00 there was a strong conjunctival hyperhemia with slight hemorrhages, intense miosis of the left eye. No changes in the right eye. The ocular tonus of the left eye was respectively 2 and 4, while that of the right one continued at 4 and 7 with the weights given above. At 11:20 the intense miosis persisted, there being already a slight edema of the cornea, conjunctival chemosis; ocular tonus of the left eye 3 - 6, the right one still at 4 - 7. At 11:40 the edema of the cornea as well as the chemosis were more pronounced. Intense subconjunctival hemorrhages. Very marked miosis with the slit lamp. We noticed that the vessels of the iris were dilated and a marked tyndallization of the aqueous. At 2 P.M. the tyndallization had disappeared, as well as the edema of the cornea. A slight chemosis persisted. Miosis disappeared and normal reflexes. The ocular tonus of the left side was 7/10 while that of the right one was 5/8.

14 — Rabbit No. 555. Ocular tonus at 10:52 A.M. 4 - 7 respectively with weights of 5.5 and 7.5. Injection at 10:53 of 2/10 cc. of a solution of the venom of *B. jararacussú* (4/10 milligrams) under the conjunctiva of the left eye after anesthesia with a ½% solution of pantocaine. At 11:07 there was intense miosis. The ocular tonus was in the left eye at 2 - 4, that of the right one remaining at 4 - 7. Slight conjunctival hyperemia at the place of injection at 11:32. The tonus of the left eye was 3 - 6, that of the right one remaining at 4 - 7. There was intense miosis, very marked chemosis and edema of the cornea, slight subconjunctival hemorrhages. At 12:45 there was a marked edema of the cornea in the left eye, intense chemosis and miosis. Some subconjunctival hemorrhages and tyndallization of the aqueous could be seen with the aid of the slit lamp. Abundant conjunctival secretion of the membranous type. At 8:30 the tyndallization had disappeared, as well as the chemosis, the edema and the miosis. Ocular tonus was 7 - 10 in the left eye, that of the right one being 5 - 8.

15 — Rabbit (not numbered). Ocular tonus at 11 in both eyes 4.5 - 7. At 11:15 subconjunctival injection of 1/10 milligram of the venom of *Crotalus terrificus* diluted in 2/10 cc. of water, in the left eye. At 11:20 the ocular tonus of the left eye was 4 - 6, while that of the right one remained 4.5 - 7. At 12 o'clock there was chemosis and slight conjunctival hemorrhages, absence of tyndallization of the aqueous and of edema of the cornea. Pupils normal with normal reflexes. At 9 P.M. there was very marked chemosis and edema of the eyelids which closed the eye and prevented the opening of the lids. Cornea, aqueous and iris normal. Tonus of the left eye 10 - 12; of the right one 4.5 - 7.

16 — Rabbit No. 532. At 10:10 we instilled two drops of fresh pure venom of *B. jararaca* in the conjunctiva and the cornea of the left eye, keeping the lids apart during thirty seconds, to make sure the venom

came in contact with the conjunctiva and cornea. At 10:26 there was a strong edema of the eyelids, intense chemosis and edema of the cornea, miosis, conjunctival hyperemia, and slight subjunctival hemorrhage. Twenty-seven hours afterwards the edema of the cornea still persisted and there was infiltration, chiefly in the exposed part of the cornea. The chemosis also persisted, with scanty secretion. Pupil immovable and of normal size. No changes in the fundus. "Milky" cornea. The time of coagulation of the rabbit's blood, after 24 hours was thirty seconds.

17 — Rabbit No. 531. At 10:12 we instilled in the left eye two drops of fresh pure venom of *Crotalus terrificus*. At 10:28 there was hardly any local reaction, only the pupils were both dilated and without reaction to light. Fundus unaltered. The time of coagulation of this rabbit, after 24 hours, was 50 seconds.

18 — Rabbit No. 558. At 10:14 we instilled into the left eye two drops of fresh pure venom of *B. jararacussú*. At 10:28 strong edema of the lids, chemosis, conjunctival hyperemia, slight subconjunctival hemorrhages, membranous secretion, miosis. Twenty-seven hours afterwards intense keratitis, principally in the part exposed. Chemosis, marked membranous secretion, edema of the lids. In the left eye there was mydriasis and absence of the light reflex. Retinal vessels dilated. The time of coagulation, after twenty-four hours, was more than two hours. Ninety-six hours afterwards, the cornea of the left eye, under examination with the slit lamp, showed deep ulceration passing completely through, and hernia of the iris. This rabbit was then given a parenteral injection of 5 milligrams of venom of *Crotalus t. terrificus*, showing one hour afterwards slight dilatations of the retinal vessels. This rabbit died two hours after injection of the *Crotalus* venom.

Doves were also inoculated with lethal doses of the venom of *B. jararaca*, *B. jararacussú*, and *C. terrificus* to verify the toxic power of the venom employed in the experiment carried out, and in these birds pupillar mydriasis was always found before death from the three venoms mentioned. From the experiment with rabbits 1 to 6, we conclude that the venom of *B. jararaca*, in sublethal doses, causes the following phenomena: blindness, edema, slight hemorrhage in the disc; ampulliform dilatation of the retinal vessels, chiefly in the terminal part, with slight hemorrhages adjacent; hyperemia of the vessels of the conjunctiva and pericornea, as well as those of the iris; pupillar mydriasis with retardation of the light reflex. All these phenomena tend to subside. There is no alteration of the ocular tonus. It is to be noted that blindness cannot be explained only by the ophthalmoscopic signs verified, since with the venom of the *B. jararacussú* these symptoms are also present without any apparent blindness.

The experiments with rabbits 7 and 9 show that the venom of the *B. jararacussú* in sublethal doses causes conjunctival, iridian and retinal hyperemia, the last two with ampulliform dilatations. Absence of retinal hemorrhages. Slight edema of the disc. Mydriasis with retardation of the light reflex. Absence of blindness. There is a tendency of the above described phenomena to subside. Absence of alteration of the ocular tonus.

The experiments with the rabbits 10 to 12 show that the venom of the *Crotalus terrificus* in lethal and sublethal doses causes pupillary modifications, characterized by temporary miosis, followed by mydriasis with retardation of the light reflex. The retinal vessels are at first dilated and then undergo a contraction, with ampulliform dilatations and slight peripheral and terminal hemorrhages. Slight edema of the optic nerve. The visual function seems to cease twenty-four hours after the inoculation. The ocular tonus does not seem to be affected by the venom.

The experiments made with rabbit 13 show that the venom of *B. jararaca*, in a dose of 4/10 milligram in a subconjunctival injection produces a local reaction of great intensity, with edema and slight hemorrhages, accompanied by immediate miosis and a marked increase of the ocular tonus; afterwards there is the formation of exudates in the aqueous. About ten hours after the injection, the inflammatory phenomena subside, and there is a marked lowering of the ocular tonus, as compared with the other eye.

The experiments with rabbit 14 show the action of the venom of *B. jararacussú*, in a subconjunctival injection reaction is less intense, chiefly as regards the hemorrhages. There was, however, a marked formation of conjunctival secretion of membranous aspect. The action on the pupil and the ocular tonus was identical to that described for the *B. jararaca*.

The subconjunctival injection of .10 milligram of the venom of *Crotalus terrificus* in rabbit 15 showed the late tonus reducing action ten hours afterward, after a slight, immediate tonus increasing action. Absence of phenomena on the side of the cornea and the uvea. Pupil unaltered. Only a strong chemosis and slight conjunctival hemorrhages. Marked edema of the lids, preventing their movements.

The instillation of pure venom of *Crotalus terrificus* in the conjunctiva in confirmation of the observation of Vital Brasil[32] showed the weak local action and the absorption of venom thus applied.

The instillation of venom of *B. jararacussú* showed the local irritant action of the venom, characterized by abundant membranous secretion. The absorption of venom, thus applied, was also shown.

The instillation of the venom of *B. jararaca*, as Vital Brasil[32] also observed, provoked phenomena of local irritation similar to those noticed with the venom of *B. jararacussú*, less intense toward the side of the cornea and without the appearance of secretion. The time of blood coagulation, after 24 hours, showed that there had been absorption of the venom.

The inoculation of lethal doses of venom in doves showed the constancy of pupillar mydriasis.

The solutions of the poison in the experiments were compared with the injections in doves in order of their toxic power. Thus a solution of 4 milligrams of venom of *B. jararaca* caused the dove's death in twenty minutes. A 0.006 milligram solution of the venom of *B. jararacussú* caused death in twenty minutes. And a 0.0015 milligram solution of the venom of *C. t. terrificus* caused the dove's death in twenty-four hours.

The fact should be emphasized that the individual resistance of rabbits to snake venom varies greatly, as appears from the fact that some died after weaker doses, while others, after stronger doses, survived; also some that received weaker doses died before others that had been given stronger ones. This fact, however, does not affect the results observed, in view of their constancy. The minimum lethal doses of the poisons which we used are, according to Vital Brasil,[31] the following: *B. jararaca*, 7 milligrams per kilo; *B. jararacussú*, 3 milligrams per kilo; *C. t. terrificus*, 1 milligram per kilo.

Koressios[33] from the experimental studies in which he observed the action of the *Naja* venom on the eyes after a parenteral application, to which we have already referred, concluded that this action was threefold: (1) the production of miosis of about a half hour's duration; (2) the provocation of an increase in the retinal blood pressure, and (3) of a passing constriction of the arteries of the fundus.

For clinical studies in which he tried to verify the action of the venom on the retina of normal and pathological individuals, with Baillart,[34] he used quantities of venom, physiologically proportioned, taking as unit the fifth part of the minimum necessary,

when injected into the femoral vein, to cause a drop in the carotid blood pressure of three millimeters of mercury in rabbits of 3 kilos. He was led to make these clinical experiments by his observation of the improvement in the sight of a patient suffering from a syndrome of encephalomyelitis in whom he tried *Naja* venom to alleviate the pain. This patient showed extensive hemorrhages in the vitreous and couldn't see to walk alone on the day after the first injection; his sight improved so that he could do so, and the improvement held for six months, when new hemorrhages reduced him to his former state.

From his experiments Koressios concluded that snake venom acts on the unstriated muscular fibers of the walls of the vessels; that the action appears in the capillary vessels at the fundus, which are at first contracted and then dilated; that in cases of capillary arteritis, venous thrombosis, and arterial spasms, it causes a rapid, marked improvement in the sight; that although organic lesions of the fundus oculi do not seem to undergo any notable modification, in some cases a marked diminution of the hemorrhages and of the edema of the disc was noticed.

Chassaing[35] in his thesis, in which he made a thorough study of the action of *Naja* venom on the eye, from a therapeutic and physiological point of view, arrives at conclusions similar to those of Koressios. Likewise Tille,[36,37] collaborating with Koressios and Chassaing, shows like results obtained in the treatment of vascular retinal diseases with that venom.

Manes[38] reports having observed an improvement in the visual acuity and visual field in a patient suffering from optic neuritis in which he applied *Naja* venom into the muscle. Also in a case of disseminated choroiditis and myopia of 18 diopters he obtained an improvement in the visual acuity with the use of this venom.

Brecher,[39] using a mixture of Kretschsky's "Immenine" and detoxicated snake venom (Vipera Verus) according to the ideas of Phisalix and Langer, obtained favorable results in the treatment of trachoma. The remedy obtained by Brecher is eighteen times less toxic than the venom from which it is derived, and seems to act on the endothelial reticula, preventing an excessive pathological proliferation of the tissues. This remedy is found on the market under the name of Trachozid. The process of preparing the venom is the secret of Brecher and had not been published. Favorable results were obtained with Trachozid in the treatment of trachoma and especially of the pannus trachomatosis by

Brecher,[40] Olah-Keppich,[41] Charamis,[42] Lobel,[43] Lodato,[44] Manolesco,[45] etc. The treatment of iritis and scleritis (Charamis) with Trachozid showed doubtful results, but it proved efficacious in the therapeutics of scrofulous keratitis. Trachozid is used in subconjunctival injections in the conjunctival fornix. It is easily tolerated and has never caused injurious effects.

Valle[46] in leprous patients with uveitis and keratitis in which pain was the predominant factor, used "anaveno crotalico," a solution of the venom of *Crotalus t. terrificus* detoxicated by formol, prepared by the Instituto Butantan of Sao Paulo, Brazil. The results, according to Valle, were always very good, the remedy being applied in subconjunctival injections, preferably in the part of the conjunctiva corresponding to the lesion, or in the four cardinal points more or less on a level with the equator of the globe. When applied by instillation the results were not favorable. He also used "anaveneno crotalico" with good results in a case of ulcer of the cornea and in a case of luetic iridocyclitis, always in subconjunctival injections to relieve pain, the other usual treatments being made at the same time.

We also used anaveneno crotalico to lower ocular tonus in accordance with the observations of Gianantoni, but without noticing, however, any effect on the ocular tonus. Correa Neto[47] used with favorable results "Soluto crotalico," which as the name indicates, is a solution of the venom of *C. terrificus* diluted 5:1,000,000, prepared in the Instituto Vital Brasil. By injecting doses that varied from 1/100 to 3/100 milligrams, he obtained a diminution of ocular algias arising from trigeminal neuralgias and inflammatory glaucoma.

Swett,[48] using a solution of the venom of *Agkistrodom piscivorous* 1:3,000, obtained very favorable results in the treatment of six cases of postoperative recessing hemorrhage. He asserts textually:

"We had the opportunity to use "Hemocoagulase," a product prepared by the Instituto de Butantan in Sao Paulo, derived from the venom of *B. jararaca*, and isolated disintegration according to the process of V. Klouzitsky and Koenig.[49] We applied it locally to clean the wound of blood in ocular operations commonly disturbed by hemorrhages, e.g., operations on the eyelids in the regions of the lacrimal sac, which had been operated previously, and we always had very good results at least equal to those obtained

with other hemostatics derived from tissues. We also used hemocoagulase in intramuscular injections on the eve of or a few hours before the operation and found the results very satisfactory. We also used this remedy in the treatment of post operative bleeding after intrabulbar operations and in retinal bleeding of several sources, always with very good results.

According to Calmette,[50] Laignel-Lavastine,[51] Taquet,[52] Castro-Escalada,[53] *Naja* venom and also others with marked proteolytic action can be used in the treatment of inoperable malignant tumors situated in the eye.

Vasquez Barriere[54] used without noticeable result a solution of snake venom in cases of malignant tumors of the orbit and in cases of vascular retinal diseases as Baillart recommends.

As we have just stated, snake venoms have been employed for various purposes in the treatment of diseases of the eye. Thus the venom of *Naja sputatrix* was recommended in the treatment of high retinal blood pressure, this indication being based on the pressure reducing effect acting as it seems directly on the walls of the vessels. Unfortunately, however, in opposition to the favorable results referred to by various authors (Vernes, Koressios, Baillart, Chassaing, etc.) are the observations of others who could not obtain similar results (Vasquez Barriere, etc.). For the treatment of intra-orbital tumors the use was suggested by analogy of the use of snake venom in intratumoral or intramuscular injections, this indication being based (Calmette, etc.) on its proteolytic action, capable of destroying the tumor directly or through substances arising from its decomposition. The use of snake venom for that purpose clashes with the fact that the selective action of the venom on the tumor cells has not been proved and there is even the danger of its stimulating the growth of the tumor, or of the formation of metastases by pieces of tissue detached precisely by the proteolytic action of the venom (v. Klobuzitzky).

In the treatment of glaucoma the use of *Naja* venom was recommended (Gianantoni) for its miotic action and that of lowering ocular tonus, but its use hasn't proved equally useful in the hands of other experimenters.

To combat ocular pain arising from inflammation of the cornea and of the uveal tract or even due to increased ocular tonus, the use has been recommended of a solution of venom of *Naja* or *Crotalus*, applied by instillation or by subconjunctival injections,

this indication being based on the neurotoxic action of these venoms. The records showing favorable results (Valle, Correa, Neto, etc.) are, in this case, more numerous than those showing less encouraging ones and it seems snake venom, by its neurotoxic action, may really be of great help in relieving pain in diseases of the eye.

In the treatment of trachoma the use of a derivative of the venom of *Vipera berus* and that of bees known as "Trachozid" is advocated. This remedy, which has proved very effective, according to various experimenters (Brecher, Charamis, Lobel, etc.), is applied in subconjunctival and intratarsal injections. But in spite of being unquestionably a useful element in the therapeutics of trachoma, it cannot, as yet, be considered a specific.

To avoid and combat bleeding during and after operations, and even in many operations, and even in ocular disease in which bleeding is frequent, use has been made of derivatives of snake venom *(B. jararaca, Notechis sculatus, Agkistrodom piscivorus, Vipera russelli, etc.)* with very good results (Swett, etc.). The indication for this treatment is based on the coagulating action of these venoms, which seem to act as thrombin, transforming fibrinogen into fibrin.

The study of the various elements found in snake venoms, whose action is not yet sufficiently known, will perhaps render possible in the future their more frequent use, and especially that of the principles which can be isolated from them. The venom, being composed of various elements, acting differently, has had its therapeutic use limited by the impossibility of applying each pharmacodynamic property separately. The weakening of the venom by heat, by formol or by neutralizing certain harmful elements, although it may have a selective action in the repression of certain undesirable elements, at the same time weakens the action which is to be utilized and retains, even though weakened, the undesirable elements.

Nothing but the isolation of the active principle, whose therapeutic property is to be utilized, will solve the problem. This is why we emphasized the value of V. Klobusitzky's work, who succeeded, together with Koenig, in isolating from the venom of *B. jararaca* the active principle which has only a coagulating effect. This isolation was obtained by the proper disintegration, and subsequent withdrawal of all albumin particles of globulin, by means

of lead acetate, and the substance which acts in very dilute solutions and extraordinarily active, belongs to the category of enzymes, being a kind of "coagulase." Recently Slotta[56] also succeeded in isolating the *neurotoxic* principle of the venom of *C. t. terrificus*, crystallizing it. This neurotoxic principle, in its crystallized state, is twice as active as the pure venom and has the double phospatidasic hemolytic and neurotoxic property. Slotta believes that this action is in fact one and the same, the venom acting accordingly both on the lipoid of the nervous tissue and on the lecithines.

This isolation of the active principle with definite pharmodynamic properties, will in days to come, encourage the use, scientifically authorized of various derivatives of snake venoms in the treatment of many diseases. For the moment, however, their use is still much restricted, limited really to a few indications.

In closing we wish to thank Dr. Harry Gradle, President of the American Academy of Ophthalmology and Otolaryngology, for his kind invitation to read this paper and for his suggestion of the subject. We also wish to thank Dr. D. von Klobusitzky for his help in our experiments, for the bibliography which he placed at our disposal, and his constant assistance in the preparation of this work. We also thank the Institute de Pinheiros for allowing us to carry out experiments in animals and for the samples of "Bothropase" which we offered our American colleagues for experimental purposes. We also wish to thank Dr. J. R. Arantes, Director of the Institute de Butantan, for the use of its library and for the venom placed at our disposal for experiments.

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THE OCCURRENCE OF VERTICAL ANOMALIES ASSOCIATED WITH CONVERGENT AND DIVERGENT ANOMALIES

A CLINICAL STUDY

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SINCE SOME anomaly of the vertically acting muscles is often associated with a lateral anomaly, a study of 11,600 cases was made in the hope of gaining some definite evidence as to the frequency of occurrence, of the condition producing the vertical anomaly and of the effect of one condition upon the other. One thousand nine hundred fifty-five of these cases showed some motor anomaly and of these 715 or 36.6% had a vertical anomaly.

Convergence excess alone, or complicated with a divergence insufficiency was found in 16.6%; 39.8% having a vertical deviation.

Divergence insufficiency alone, or complicated by a secondary convergence excess was found in 2.5%; with 59% having a vertical deviation.

Divergence excess alone, or complicated by a secondary convergence insufficiency was found in 7.5%; with 59.9% having a vertical deviation.

Convergence insufficiency with a secondary divergence excess was found in 2.8%; 40% having a vertical deviation.

It was not thought practical to attempt to state the great variations in the amounts of hyperphoria and hypertropia. These varied from 1^{Δ} to 55^{Δ}.

The cause of the underaction of the vertically acting muscles could not be determined in many cases — that is, whether the muscle action was limited because of the structure, insertion or innervation of the muscles; likewise whether the overaction was

due to a primary spasm of the muscle or to a secondary deviation of the associate muscle or to a secondary contracture of the direct antagonist. Again, it is a generally accepted theory that the act of sursumvergence is analogous to that of convergence and divergence and undoubtedly some cases observed, especially in the higher degrees of imbalance had some overaction of sursumvergence.

An underaction more or less marked of the vertically acting muscles, was found in the following order:

Superior rectus	Unilateral	347
	Bilateral	160
Inferior rectus	Unilateral	99
	Bilateral	22
Inferior oblique	Unilateral	17
	Bilateral	3
Superior oblique	Unilateral	12
	Bilateral	1

The hyperphoria was unclassified in 44 cases. Cases of primary spasm were very rare in our opinion. Other observers claim a much higher percentage of primary spasms but this is a controversial subject. But few primary spasms of sursumvergence were observed. Where the function of sursumvergence was excessive there were but few cases where the vertical imbalance did not increase definitely in some field. This could often be demonstrated by diplopia tests but more accurately by the screen and the screen-comitance test in the six cardinal fields. This ruled out a primary anomaly of sursumvergence.

In the 1,955 cases, 873 has an uncomplicated convergence insufficiency and 20 were retraction syndromes, or other like congenital anomalies. This leaves 1,062 cases divided as follows:

Uncomplicated lateral deviation	347
Uncomplicated vertical deviation	358
Combined vertical and lateral deviations	357

Thus two in every three of this group, or two in every five of the 1,935 cases had a vertical imbalance.

Of the 1,062 cases, 338 had a refractive error of ½D or less and of these 29% were of the plain lateral group, 47% were of the plain vertical group and 23% were of the combined lateral and vertical group. Two hundered eighty-six cases had a hyper-

metropia of 2D or less and of these 36% were of the plain lateral group, 34% were of the plain vertical group and 30% were of the combined lateral and vertical group. Two hundred five cases had a hypermetropia of more than 2D, and of these 50% were of the plain lateral group, 14% were of the plain vertical group and 36% were of the combined lateral and vertical group. The remainder were myopic, and were about evenly divided among the three groups.

Of the 1,062 cases, 767 had a visual acuity of 20/25, or better, in each eye and of these 28% were of the plain lateral group, 41% were of the plain vertical group and 31% were of the combined vertical and lateral group. Fifty-four cases had a visual acuity of 20/25 or better in one eye, and 20/40 or better in the other eye. Of these 41% were of the plain lateral group, 16% were of the plain vertical group and 43% were of the combined lateral and vertical. Eighty-one cases had a visual acuity of 20/25 or better in one eye and less than 20/40 in the other eye. Of these 63% were of the plain lateral group, 14% were of the plain vertical group and 23% were of the combined lateral and vertical group.

The tests used were the screen (cover) test, combined when practical, with the parallax test. This test was made routinely at 6 m., 33 cm. and in the six cardinal fields at 33 to 40 cm. In cases of divergence excess and insufficiency, the distance was often increased up to 150 feet, while in convergence excess and insufficiency, the test object was brought in to 20 cm.

In the phorias and the lower degrees of tropias, the Maddox rod or the combined screen-Maddox rod tests were used with the screen-parallax to check one against the other.

Diplopia was formerly routinely plotted on the tangent curtain, but the field of its use is quite limited, since it cannot be used in young children, in cases of suppression or amblyopia, nor in cases having a large deviation. The tests before mentioned have been found to be much more reliable. Other tests were employed as confirmatory when desired.

Andrew Lang has said, that "statistics are as a lamp post to a drunk — used for support rather than illumination." Having no theory to support, it might be well to get what illumination we can from a review of some of the cases as representatives of the various anomalies. The conclusions are drawn partly from the cases included in the statistics, but largely from the cases going through the muscle clinic of the New York Post Graduate School

and Hospital, the monthly visits to the Brooklyn Eye and Ear Muscle Clinic, or cases seen with confreres in many different cities. These of course are not included in the statistics.

Little, if any correction is obtained from plus lenses, even where the vertical error is small. Many such cases had been wearing low plus lenses for some years with no improvement.

In higher degrees of hypermetropia with squints ranging from 40 to 70$^{\Delta}$, usually only 10 to 20$^{\Delta}$ was corrected by lenses if a *vertical imbalance* were present. Two cases treated at the New York Post Graduate Muscle Clinic recently, illustrate this.

The first case, a girl of ten with a hypermetropia of 4.5D, had 50$^{\Delta}$ of convergent squint, with no vertical imbalance. The esotropia was reduced to an esophoria of 5$^{\Delta}$ by full correction. The second case, a boy of the same age, with a hypermetropia of 4D, had an esotropia of 55$^{\Delta}$, with a vertical imbalance. Full correction reduced the esotropia to 40$^{\Delta}$, correcting but 15$^{\Delta}$ of the lateral deviation. These cases are very typical of the effect of plus lenses, in the absence or presence of a vertical anomaly. However, the *unusual* case with a vertical imbalance is converted by glasses alone, into a divergent squint of approximately an equal amount.

Convergence insufficiency or divergence excess whether simple or combined, are usually made worse by the use of plus spheres. Occasionally it is necessary to withhold both spherical and cylindrical correction unless they relieve symptoms of headache or asthenopia.

Pure divergence insufficiency is unaffected by the use of lenses. However, if a convergence excess is superadded, then plus lenses may relieve the symptoms due to the convergence excess, while the deviation for distance remains practically unchanged.

Myopes with a convergence insufficiency or a divergence excess, are frequently helped in overcoming the lateral deviation by a full or overcorrecting myopic lens.

In all cases the vertical error should be corrected by prisms when possible. This will depend on the degree of comitancy of the vertical deviation and the degree of comfort experienced.

Orthoptic training gives quite varying results. Many will show third grade fusion when on the test, but, at all other times, have a tropia. Rarely is the amount of hypertropia reduced by such training when measured by the screen test, but sursumvergence may be increased sufficiently to enable the vertical error to be overcome at least when concentrating.

Hypertropia in the primary position may be overcome by turning or tilting of the head and this in turn may correct the esotropia or exotropia. This result is not infrequent, and when the tilt is not marked, may contra-indicate surgical treatment and even prisms may not be well borne. However, if the head tilt is a disfigurement or produces symptoms, it is in these cases that a satisfactory result can be expected.

The following cases have been selected to show the various results seen when a vertical imbalance is present.

Case 1. — Girl, age 7, vision 6/6 right and left. Hypermetropia + 0.25 cyl. 90° OU. In September, 1934, she had a right hyperphoria of 5△, with no lateral deviation for distance. For near, there was an esotropia of 15△, with a right hypertropia of 5△. One year later, she had changed to a right exotropia of 20△, with a right hypertropia of 6△ for distance while for near she had no lateral deviation and a right hyperphoria of 6△. She fuses with depth but orthoptic training has failed to make her want to fuse, except when using the instruments, or when using the eyes in near vision. In September, 1937, the exotropia was 27△, right hypertropia 5△ for distance — exophoria 6△, right hyperphoria 5△ for near. Operation has been advised.

Case 2. — Girl, age 9, had a marked divergent squint for distance. For near, she varied from an exophoria to an exotropia. Convergence training improved the convergence near point. A recession of one externus reduced the deviation greatly and convergence exercises have been continued. When seen in September, 1938, she had an exotropia of 20△ for distance, changing to an exophoria for near. Convergence near point 55 mm.

Case 3. — Boy, age 2½, had an exotropia of 35△ for distance, with a right hypertropia. His convergence near point was normal, but poorly held. He was treated for one year with no improvement. A recession of the right externus resulted in an overcorrection. This was improved by correction of the hyperopic astigmatism. Later the right externus was resected and the inferior oblique tenotomized at the insertion with a satisfactory result.

Case 4. — J. S., age 6. Eyes crossed at 3 years of age. Full correction of + 1.50 spherical had been used with no effect. He was

operated in May, 1938. The scars indicate that the right externus had been resected and the internus receded. The scars look as if the surgical technique had been excellent. I have no knowledge of the amount of squint before operation. His vision is 20/25 + right and left, fixing right usually for distance and near. Exotropia 34$^{\Delta}$ for distance and 38$^{\Delta}$ for near. Relative convergence near point 120 mm., left eye diverging. Right hypertropia in the primary position of 5$^{\Delta}$, which increases in looking up to the left. In the upper right field, there is a left hypertropia. Converging exercises have had little effect. Such an overcorrection is not unusual, when a vertical imbalance is present. A recession of the external rectus of the diverging eye with a tenotomy of the inferior oblique at the insertion gave an excellent cosmetic result.

Cases 5 and 6. — Brother and sister operated by an excellent surgeon for convergent squint. In each case a unilateral recession was chosen. One resulted in a divergent squint for distance and near, both with and without glasses, while the other was divergent with glasses for both distance and near, and without glasses, diverged for distance only. They each had a vertical imbalance.

Case 7. — Man had a divergent strabismus of 40-50$^{\Delta}$. Both externi were receded. This resulted in a convergent squint of a similar amount. The operator then advanced the left externus, resulting in a divergent squint of 40$^{\Delta}$. There was a left hypertropia of 20$^{\Delta}$ present, due to a right superior rectus paralysis. The vertical imbalance without doubt was responsible for the wide lateral swing of 80$^{\Delta}$ and back.

Time does not permit to multiply cases which give strong evidence that a vertical imbalance is an active factor in producing convergent and divergent strabismus. This also seems responsible for the unsatisfactory results from treatment by glasses, orthoptic training, or operation.

If the divergence in a surgically overcorrected convergence strabismus, or the convergence in an overcorrected divergence strabismus were due to a secondary deviation from fixing with an eye in which a muscle had been receded or tenotomized, then it should be found as frequently if no vertical imbalance were present. The fact that practically all such results have a vertical imbalance and that these failures are those of many different and excellent operators, would seem quite definite proof that the overcorrection is not

due to the new adjustment of the lateral muscles, but rather to the vertical imbalance. The vertical imbalance in these cases seems definitely responsible for the unsatisfactory results from treatment by glasses, orthoptic training or operation.

SUMMARY

It is significant:

That those cases, which did not show definite improvement from lenses or means other than surgery with few exceptions, had a vertical imbalance.

That in the failure from surgical treatment either of overcorrection or of undercorrection in our own cases, or in those seen at the various places stated, there has been almost without exception, a vertical imbalance.

That the operative failures of many excellent surgeons have been examined. These cases were both of convergent and divergent squints. The choice of operation varied greatly, depending upon the preference of the surgeon. With almost no exception, a vertical imbalance was found to be present.

That in uncomplicated convergence excess with either a lower or higher degree of hypermetropia, a double recession will give a most satisfactory result. However, when a vertical imbalance is present, the same amount of recession for an equal amount of lateral squint may result in undercorrection occasionally, but more frequently in an overcorrection, about as much as the original squint. This result may be delayed, but may develop immediately after the operation.

That in uncomplicated divergence excess with a normal convergence near point, a conservative recession of one or both externi gives a good result. In the presence of a vertical imbalance, there may be an undercorrection, but much more probably a marked overcorrection.

That in a convergence insufficiency with a secondary divergence excess or when the divergence excess is primary, one can judge quite accurately of the operative results when there is no vertical imbalance. But in the presence of a complicating hypertropia, the same amount of recession or resection may result in a definite overcorrection, but much more probably to an undercorrection of the lateral deviation.

That these failures become less if the vertical imbalance can be corrected before the lateral. When the lateral imbalance is very marked it may not be possible to properly study the vertical. In such cases the lateral deviation may be corrected first, holding the correction of the vertical deviation until later, when it can be more accurately studied. Quite rarely the correction of the lateral will lessen the vertical deviation. Not infrequently, especially in a unilateral strabismus, the lateral and vertical deviations can be satisfactorily operated at the same time. The most frequent of this group is for example, in a unilateral right convergent or divergent strabismus, associated with an overacting inferior oblique of the same eye, which definitely should be operated. When the external rectus is in the grasp of the muscle forceps and has been tenotomized, the inferior oblique may be tenotomized at the insertion, after which resection or recession of the externus is continued. For lesser amounts of inferior oblique overaction, such a procedure as the Blaskovics should be considered. The inferior oblique was receded in one case with very satisfactory results.

CONCLUSIONS

1. Vertical anomalies are a frequent complication of lateral deviations.

2. No case of lateral strabismus should be operated or otherwise treated without a thorough study and analysis of the vertical element when present.

3. When operating to correct a lateral deviation, it must be remembered that the rules of the game are not the same if a vertical factor is present.

4. In cases of convergent strabismus in which it is thought best not to correct the vertical deviation first, we believe there is less danger of an overcorrection if the first procedure is a resection of one or both externi, leaving a recession of the interni for a finer adjustment later if desired.

5. The more nearly one evaluates the several elements in these complicated cases, the greater will be the percentage of success.

DISCUSSION

DR. ALBERT C. SNELL, Rochester, N. Y.: Whenever Dr. White presents a paper relating to muscle anomalies, I am sure that we all stop, look and listen, for we are very sure we will gain some practical knowledge from his paper. This present paper of Dr. White's is no exception, with his very carefully gathered statistics and the deductions which he has so carefully made.

I have not, myself, compiled any great amount of data but in the study of the muscle anomalies in a thousand cases of myopia which I reported in the Transactions of the Section on Ophthalmology of the American Medical Association, I found that convergence insufficiency was rare, its frequency being four per cent (that is confining my statistics entirely to myopic cases); that hyperphoria associated either with exophoria or esophoria had an incidence of about five per cent; that divergence excess and esotropia were common. However, this latter condition was usually found to be associated with abnormal visual acuity, at least in one eye. In other words, in these cases of divergence excess in myopia I felt that the myopia was probably not the cause for the divergence excess because I have records of a number of cases which originally were far-sighted and had a divergence excess and became myopic.

One constantly sees, of course, some patients with low degrees of myopia who have persistent convergent strabismus, but it is still more common to find divergence with myopia. But as I said, it seems to me that we haven't yet been able to establish the relation of cause and effect in this class of cases. I am under the impression that divergent excess is probably present in a very large percentage of cases of progressive myopia.

In the cases of myopia, I found convergence insufficiency in about one to four per cent, and a subnormal amplitude present in only one per cent. In a large proportion of all cases of lateral deviation, other factors than ametropia are present: for example, anisometropia, convergence and divergence excess, vertical deviation and defective fusion sense. Convergence insufficiency is found associated with hypermetropia much more often than with myopia in the ratio of one to eight in a review of a limited number of cases which I went over recently. Convergence excess is more often found associated with myopia than with hypermetropia in the ratio of one to three.

I think that observation probably doesn't agree with our general impression, but statistics which I have gathered seems to bear that out, and I was surprised, myself, to find that observation true. It would seem to me that if some of these observations are correct, they strongly indicate that the condition of refraction is not of primary or fundamental importance in the causation of myopia or in the status of the muscle anomolies in these cases.

I am in agreement with Dr. White that a large number of cases of exophoria begin as a convergence insufficiency, especially when a vertical deviation is present. When there is present a vertical deviation, 8 prism degree or more associated with a divergence, an operation to correct this deviation is usually indicated and as a general rule should be performed at

an early date. Of course, I am entirely in agreement with Dr. White that where we have the vertical devition combined with other phorias or tropias, the handling of the vertical deviation is extremely important.

A careful examination must be made of the action of all possible muscles concerned, especially keeping in mind the action of the oblique muscles to determine the proper operative procedure. In my experience, without surgical correction of the vertical deviation other forms of treatment are rarely successful. After correction, non-surgical treatment is indicated and often successfully overcomes the lateral deviation when fusion is established.

In a certain number of these cases I think we should look out for the nervous element, as Dr. Lancaster pointed out. I have had several cases under observation in the last few months which were probably the highly neurotic type, and it is my impression that where we find the muscle deviation inconstant in more than one examination, or during the time of the examination, there is very apt to be present in those cases a decided nervous element. I think when we consider any operative procedure, we should be pretty much convinced as to the presence or absence of some form of nervous disturbance in cases that are highly neurotic.

Occasionally, of course, we run across a very unusual sort of case. Recently I had under observation a young man who was a student at Harvard Medical School for one year, and was an honor student. He had to discontinue his studies because of his eyes. He had a prescription with a +.50 sphere in one eye and +.75 in the other, and under mild cycloplegic I found two and a half diopters in one eye and three in the other, with slight astigmatism. In a case like that, I think most of us would immediately suspect that patient of having probably a divergence excess, but this young man had a very distinct convergence insufficiency. He had a convergence near point of about 150 and he was not able to maintain near vision. With the amount of hypertropia which he had, he was able to see at a distance but he wouldn't accept the correction for distance. He apparently had sort of a spasm of his accommodation, because he would overcome his handicap for distance and get along very well, but he was unable to do any reading, and he enjoyed having the full correction for all his close work, and combined with prisms base in, he has been able to continue with his work quite easily.

I think the indications and rules and methods of procedure for treating these more complicated cases have been presented by Dr. White in a most instructive and valuable manner. In all cases of muscle deviation, one should acquire all possible information in regard to the many complex factors which may be present in any given case in order to determine the proper treatment, especially that needing operation. Dr. White's many contributions in this field are a most valuable aid.

Dr. P. Chalmers Jameson, Brooklyn: I think Dr. White has given us a very valuable résumé of the conditions which he outlines. There is no man more capable of analyzing the vertical anomalies and their influence upon lateral deviation than he, nor is there anyone who has a larger number of these types under command.

The task of analyzing 11,603 cases and finding therein 1,900-odd anomalies, with 715 vertical anomalies, is no small one, but when accompanied with the incidence of excess and insufficiency, the incidence of refraction as bearing upon the anomaly, the place of orthoptic training, the task assumes colossal proportions.

As I see it, the theme of Dr. White's paper is the erratic return of correction after operation. Dr. White has dwelt upon the question of computation, as Dr. Snell so ably did later. Computation is a very important matter if one is going to get the best out of muscle work, and it has been considered very much more in recent days than formerly. We used to take deviation alone and go ahead and operate, and trust to luck, but we don't do that now.

Out of Dr. White's 1,200 cases in which there were no vertical deviations, I would like to observe that in these cases there would probably be, after operation, an erratic return of correction of eight or ten per cent.

The computation which is so essential, to which Dr. White called our attention, includes so much: muscle excess, muscle insufficiency, palsies, secondary contraction, secondary deviations, muscle attenuations, muscle hypertrophies. How often, after exposure, we do find a large hypertrophied muscle! I remember when reading a little paper in London at one of the congresses there, when I described one of these large congenital muscles, a very eminent muscle surgeon was heard to whisper to his associate, "Those muscles don't exist."

Now they do exist. A little later in my own operating room I picked up a large muscle and called Dr. Reynolds' attention to it. Dr. Reynolds was one of the wittiest of men. "Dr. Reynolds," I said, "how about this for a large muscle?"

He promptly answered, "That is not an ocular muscle; it is a gluteal muscle."

These muscles are very large at times. This point I want to make, that in coming to a conclusion as to how we are going to avoid these erratic results, we obtain much information by the exposure of our anatomical field. Tenotomies used to be all done in the dark and we did not expose our field and therefore we frequently did not know what we were operating upon. The field should always be exposed in order to ascertain the anatomical conditions which exist. If we don't practice this, we are working in the dark. We can ascertain so much also about the fascial conditions.

Dr. White has spoken about the retraction syndromes, i.e., the fascial conditions. There are many modifications of these conditions. After exposure we can pick up a capsule, can ascertain its tension, and in lateral deviations can do a vertical capsulotomy, releasing the tension if it is very great. After exposure we can make a diagnosis of hypertrophy or attenuation. Frequently the muscle on the deviating side is attenuated. We may have contemplated a Recession, but in this instance should close the wound and do an Advancement on the opposite side.

No operator should be discouraged. We may be chagrined but should never feel humiliated when we get an untoward result in muscle work. There are so many considerations which tend to defeat uniform correction that we are all bound to get a percentage of erratic return of correction.

I think Dr. White's paper is very valuable. There is no question but that he makes the point that in the future we have got to take into consideration the vertical deviations, if we are going to get the best possible correction.

Dr. White has touched very lightly on the cause of this evident fact. I suppose time did not permit of that, and then there are so many theories than can be advanced. Dr. Duane's theory, I think, touches it about as closely as any. Dr. Duane says, "A condition that favors the development of a lateral squint is the presence of a congenital vertical deviation, especially a paralysis of the superior rectus. The patient in this case being unable to fuse the double images on account of the difference in level, tries to obviate the confusion by either tilting the head or separating the images as far as possible. This he does by diverging or converging the eyes."

Dr. Duane adds, "That this is the cause of the lateral squint in these cases is proved by the fact that in some of them, particularly before the lateral deviation has become inveterate, the lateral disappears when the vertical deviation is relieved by operation."

This is the gist of Dr. White's paper.

He has made many valuable contributions to muscle work. To my mind, this is not the least valuable, and this paper may well be considered a standard for future reference.

Dr. James W. White, in closing: I wish to thank the discussers for their very kindly discussion.

Answering Dr. Snell's remark in regard to convergence insufficiency, undoubtedly he combines all convergence insufficiencies in one group. Duane's percentage in working up that article on convergence insufficiency back in 1913 or '14, I believe gave 8 per cent. That is simply my memory, but I think he gave 8 per cent of convergence insufficiency.

He classified as convergence insufficiencies, as you all know, accommodative and non-accommodative, but I have found in teaching work that the plainer you can get your English, the better you get it across, and I have been classifying my convergence insufficiencies in the last few years into the ocular and systemic. In the ocular convergence insufficiencies you find a higher percentage of vertical deviations. In ocular convergency insufficiencies you have as their cause, first, hypermetropia; next, myopia — not in that order, possibly; then anisometropia, vertical deviations, and then fusion defects, etc.

The systemic convergence insufficiencies will very commonly measure, so far as their ocular balance is concerned, practically normal. They will, however, have a remote near point of convergence and a very large percentage of them have a subnormal accommodation.

I have one patient 26 years of age with very marked symptoms, with a near point accommodation of just over three diopters. I gave him at the age of 26 a fused bifocal of plus one and a half D and he is doing his close work with a great deal of comfort with that. But going back into his history, I find that he had a streptococcus infection a few years before. You will find intestinal infections, streptococcus infections one place or

another, are a contributing cause, the muscle balance being way out of proportion to the symptoms. Muscle balance may be practically normal and symptoms may be most marked.

Dr. Snell finds that myopes are about three to one higher per cent. My figures are not quite the same as that, but I think that is just a matter of detail which doesn't mean very much and we should take them as they come.

In the case of the student with the two and a half diopters in one eye and plus three in the other, I think without any question of doubt that man had a systemic convergence insufficiency. He had a subnormal accommodation. He was in college, so he was somewhere around twenty, and the fact that he is so comfortable now with full correction, which should have been put on him early, indicates that. Of all the mistakes I find in seeing cases in consultation, the most common thing to be overlooked is a subnormal accommodation. I can't emphasize too strongly the study of accommodation when quite a large percentage of cases have systemic convergence insufficiency associated with a subnormal accommodation.

Dr. Jameson brought up the question of how to avoid mistakes. We have gone into that somewhat. Dr. Jameson has told us more about it. I did touch lightly on the cause, because this paper couldn't go into the cause, and on the other hand, in many of these cases we haven't any proof of the cause. As I stated in the paper, we don't know whether the under-action of these muscles is because of their structure, their insertion, or their innervation, and there is no way to prove those things unless you dissect them pretty well, probably test them with electric current, feel them over and look them over, or get a biopsy, and that of course you can't do because that is much worse than the condition they had to start with.

The same with the over-acting muscles. In many of these cases you can't furnish absolute proof of why you have the condition, but you measure it and find that you have this condition and you try your best to relieve it in one way or another.

I thank the President and Secretary for the privilege of presenting this paper.

TRANSPLANTATION (IMPLANTATION) OF THE LACRIMAL SAC IN CHRONIC DACRYOCYSTITIS

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THE VARIOUS methods of treating chronic dacryocystitis by the creation of an artificial passage into the nose have been reviewed extensively within recent years. Noteworthy in this respect are the publications of Chandler[1] and of Kaleff,[2] in which the advantages and disadvantages of the modifications of the Toti operation (Dacryocystorhinostomy) are fully discussed. These operative procedures can properly be classified into the following four groups, all being external in type:

Group 1. In which the anterior and posterior flaps of nasal mucous membrane and lacrimal sac respectively are sutured together according to the methods of Ohm[3] and Depuy-Dutemps-Borguet.[4]

Group 2. In this group one or more flaps of nasal mucous membrane are sutured to the anterior or posterior wall of the sac, but without forming an exact or complete anastomosis (Basterra,[5] Kuhnt[6]).

Group 3. The lacrimal sac is removed, leaving a small piece immediately surrounding the opening of the canaliculi to empty into a large opening into the nose. Sutures are not used to unite the mucous membrane of the nose with the portion of the sac (Blascovicz,[7] Hoette,[8] and others).

Group 4. Transplantation or implantation of the lower end of the sac into and through a new opening made into the nose.

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Presented by Dr. Harold Gifford.

REVIEW OF THE LITERATURE OF TRANSPLANTING OR IMPLANTING THE LOWER END OF THE TEAR SAC INTO THE NOSE

In 1904* F. Speciale,[9] working in the clinic of Professor Cirincione in Italy, performed the following operation on the cadaver: By means of a skin incision the tear sac was exposed and was freed from the surrounding tissue at its lower portion. After cutting the sac transversely as far down in the bony canal as possible, the tear sac was elevated and a 9 millimeter opening was drilled through the nasal process of the superior maxilla. The mucous membrane of the nose covering the artificial opening was incised and the lower free portion of the tear sac was inserted through the bony opening. Subsequently the operation was attempted on two patients both having a chronic obstructive dacryocystitis. In the first patient sutures were not used to hold the sac in place but in the second the circumference of the sac was sutured to the incised mucous membrane within the nose. The results of this operative procedure, however, were not satisfactory — necessitating extirpation of the tear sac in both instances. In the same year (1904) Toti[10] published his article on Dacryocystorhinostomy and Toti's operation was adopted in Cirincione's clinic.

In 1911, Forsmark[11] in Sweden described a somewhat similar operation. The tear sac was freed from its surrounding tissue, the lower part isolated and cut as low down as possible. Following this a comparatively large defect was made with a chisel through the lacrimal bone into the middle meatus of the nose. The proximal end of the tear sac anchored with a thread was pulled into the nasal cavity. The thread was brought out through the nostril and fastened to the cheek. This operation was performed on 29 patients. Of these all observed over a period of a year or more, only 50 per cent had a good result in terms of symptoms.

Neumayer[12] reported an operation from Germany in 1911, the technique of which seems to be identical to the one employed by Forsmark. Neumayer does not give a full account of his methods but states that his results were "satisfactory."

In 1920 F. E. Burch[13] of St. Paul described a method published in the Transactions of the Academy in which he freed the lacrimal duct from its bony canal downward for a distance of 5 or 6 mm. The lacrimal crest was removed with a burr or biting forceps and a bony window was made also with a burr or a 5 mm. dental

* Speciale's article was published in the year 1913.

trephine through the nasal process of the superior maxilla. The lacrimal duct, previously lifted from its canal, was slit on its nasal side from its lower end upward a short distance into the sac; the duct and sac were tucked through the opening in the bone. A soft rubber catheter or a lead stylet introduced through the canaliculus aided in holding the duct and sac in place until healing took place. The operation as described was done only three times and the opening remained patent in two instances.

J. H. MacMillan[14] of Montreal, Canada, reporting on a new operation for the treatment of lacrimal obstruction in 1921, made the opening into the nose through the lacrimal bone so that the bony defect appeared beneath the middle turbinate on the nasal side. The lacrimal sac was then drawn down through this opening by means of sutures attached to the lower end of the sac. By tying the two ends of the suture over a piece of gauze inserted into the nostril the sac was held in place in its new location. In 1932 MacMillan[15] gave the results of 25 operations so performed. Of these 17 were completely cured (no tearing and no discharge), 4 were improved with little or no discharge but with tearing and no patency with the syringe except after probing, 3 were failures and from one there was no response to inquiry.

Reports of operations in which the lower end of the lacrimal sac was transplanted into a window made into the nasal cavity were also given by Fazakas[16] in Hungary in 1924, by De Lieto Vollaro[17] of Milan, Italy, in 1929; Del Barrio[18] of Seville, Spain, in 1929; Rosengren[19] of Lund, Sweden, in 1931 and by Stock[20] of the University of Tubingen, Germany, in 1934.

It is of interest to note that of the four cases reported by Fazakas, all operations were successful. He advised making the bony opening through the ascending process of the maxilla where possible. Del Barrio, employing the technique advocated by Fazakas, reported four operations, two of which were successful at the end of ten and fourteen months' observation. De Lieto Vollaro transplanted the tear sac through an opening made at the floor of the lacrimal fossa perforating the lacrimal bone. This author at first used sutures uniting the wall of the sac and the periosteum surrounding the nasal opening but later suspended the sac in the nasal opening. Apparently his results were satisfactory in a few cases observed over a period of ten years. Rosengren reported his experience with the technique advocated by Burch and of the 10 cases operated all were successful. Stock cites the

results of 45 cases, 40 of which were followed over a period of four years — there were 6 failures. Instead of perforating the lacrimal bone with a chisel Stock used a 10 mm. trephine.

DISCUSSION

The results of this operation as performed by the various authors are difficult to evaluate. The successful maintenance of a patent passage from the lacrimal sac to the nasal cavity has been reported as varying from 50 to 100 per cent in cases where transplantation was performed. In most cases in which the lower end of the sac was implanted into an opening made through the ascending process of the maxilla, the percentage of successes have run higher to those in which the perforation was made through the floor of the lacrimal fossa. The anatomical relationship of the parts therefore seems to be an important consideration.

The lacrimal fossa is about 14 mm. in height and 6 - 7 mm. across. It is divided vertically by a line corresponding to the junction of the maxilla with the anterior edge of the lacrimal bone and each of the two bones contribute approximately an equal part in its formation. However, there are many variations (Whitnall,[21] Thorsch[22] and Fazakas[23]). The nasal process of the maxilla, forming about the anterior half of the fossa, is thick and strong, and becomes even thicker toward the anterior lacrimal crest, which curves downward and outward into the lower edge of the orbit, overlapping the lower end of the fossa in front. The lacrimal bone forming the posterior half of the fossa is thin but it thickens slightly toward the posterior lacrimal crest. The upper half of the fossa and the lacrimal bone is in relation to the ethmoidal air cells. In general, the line of the posterior crest may be taken as the posterior limit of entry into the nasal cavity. Within the nose, the anterior inferior end of the middle turbinate overlies the area of the lacrimal fossa. While all these features vary in different subjects, they show that the portion of the fossa directly related to the nose is limited to its lower half, behind the anterior lacrimal crest, an area measuring about 6 to 8mm. in diameter. A satisfactory trepanation of the bone, in order to gain free access to the nose, must therefore extend downward and forward over the lower portion of the ascending process of the maxilla. The lower half of this bone is dense and contains no air cells.

In order to approach the nasal cavity by this avenue the anatomical and pathological variation of the nose must be con-

sidered; i.e., a high deviation of the septum causing obstruction must be corrected and the anterior tip of the middle turbinate if large and overhanging the area of trepanation must be removed by a preliminary operation.

INDICATIONS

Mosher's[24] recommendation of the dacryocystorhinostomy has been a very important influence in the adoption of the external operation in this country. It would seem that the only objection to the external approach is the resulting scar. This minor cosmetic blemish is negligible when it is understood by both the patient and surgeon that the external procedure affords a better view of the operative field, easier access to the nasal fossa through a more logical site and much less trauma to the intranasal structures. There are those ophthalmologists, who while admitting that preservation or restoration of function is the surgical ideal, still prefer total extirpation of the lacrimal sac to any reconstructive procedure. Extirpation of the sac is indicated only in conditions where reconstructive operations are contraindicated, as in trachoma, where cicatrical obstructions of the canaliculi and of the sac may occur, in tuberculous processes of the tear passages and in malignancies of this region. Intraocular operations when postponed because of dacryocystitis should be preceded by an extirpation, unless it is possible to allow a few weeks to intervene between the two operations. The reason for this is clear. In purulent infections of the cornea where there is constant reinfection from the sac, extirpation is desirable and in most cases imperative.

I feel that the ophthalmologists should be thoroughly familiar with the various modifications of the external dacryocystorhinostomy operation. Each modification has its proponents but the question resolves itself into what method in a given case will give the best results with the least effort of execution. In lacrimal sac surgery, as in all other surgical procedures, much depends on the skill and experience of the operator. An ophthalmologist limiting himself to that specialty must, of necessity, avail himself of the services of an experienced rhinologist. It is of importance that before deciding on any external operation for the relief of chronic dacryocystitis a careful examination of the nasal passages be made. As a preliminary step any obstruction to a free communication must be corrected in every case; i.e., high deviations of the

nasal septum must be resected, the anterior tip of the middle turbinate should be removed if so indicated, polypoid growths removed and any sinus disease controlled so far as possible. Many of the failures following efforts at dacryocystorhinostomy are not primarily due to any technical difficulties or faults of the operation, but are due to neglect in dealing with pre-existing patholgic conditions of the nose and para-nasal sinuses.

There is no question, that conservative treatment with lacrimal probes and syringes will sometimes cure cases of early dacryocystitis in which stenosis is slight and in which a thickening of the sac wall has not occurred. But treatment by means of probes should cease soon after a few attempts have been found to be ineffective. The slitting of the canaliculus for the purpose of passing probes does not recommend itself because it tends to destroy the natural capillary and suction mechanism at the proximal end. A slit canaliculus may nullify the otherwise good result of a dacryocystorhinostomy operation. (See case reports.)

The final treatment of all cases of chronic dacryocystitis with obstruction in the bony canal and mucocele formation must be operative. The decision as to what method to use rests on the location and degree of obstruction encountered, the condition of the lacrimal sac itself, the presence of disease in the surrounding tissues and the existence of any nasal pathology.

TECHNIQUE OF IMPLANTATION OF LOWER END OF TEAR SAC INTO THE NOSE

Anesthesia

All operations except those in children are done under local anesthesia in order to give a drier operative field. Anesthesia is secured as follows: Before the operation the patient is given morphin ¼ to 1/8 grain, and scopolamin 1/200 to 1/400 grain, depending on the age and weight of the patient. The nose is anesthetized with cocain and adrenalin packs, care being taken to place the packing well up to the level of the sac. A 1 per cent novocain with adrenalin solution is injected under the skin along the line of incision and also subperiosteally in this region. About 2 ccs. of novocain are also injected close to the bone and well back along the inner orbital wall above, to block the anterior ethmoidal nerve and branches of the infratrochlear and supratrochlear nerves. Another injection is made in the region of the infra-orbital foramen (Fig. 1). This procedure gives practically perfect

anesthesia. If ether anesthesia is used the cornea should be carefully protected during the operation. If local anesthesia is employed the eye is not cocainized so that the patient will keep the eyelids closed or will wink frequently enough to prevent drying. The lower canaliculus is now dilated sufficiently with a conical probe to allow the passage of a canula and the lacrimal sac is syringed with normal saline.

Incision

The incision is placed halfway between the inner canthus of the eye and the side of the nose at least 12 mm. from the inner canthus (Mosher). It begins on a level with the summit of the globe and is carried downward in a nearly straight line, 1½ to 2 cm. in length. The incision is made down to the bone throughout its whole length. Retractors are inserted and held by an assistant who also checks any bleeding by means of a small suction canula (Figs. 1 and 2).

Fig. 1

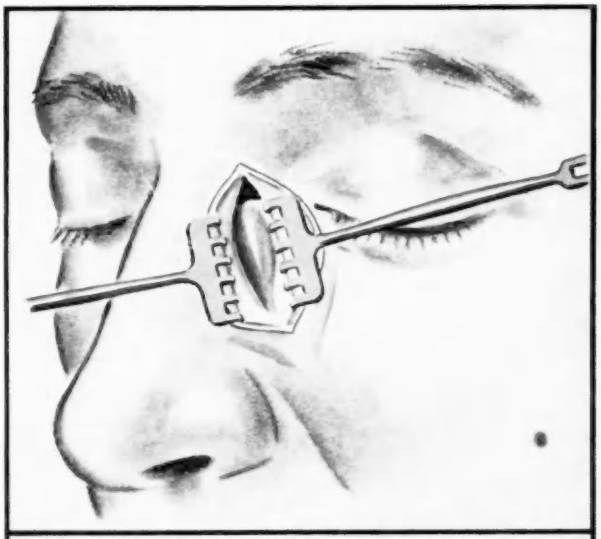

Fig. 2

Elevation of the periosteum and exposure of the sac

With a small flat chisel the bone is exposed over the ascending process of the maxilla toward the anterior lacrimal crest. With a dissector, the edge of the anterior lacrimal crest is now defined and the attachment of the anterior limb of the "Y" ligament is divided exposing the sac (Fig. 2). The periosteum lining the fossa together with the sac, is now pushed outward exposing the surface of the fossa almost as far back as the posterior lacrimal crest. The periosteum is separated downward as far as possible into the mouth of the naso-lacrimal canal. The exposed fossa is now carefully inspected for the presence or absence of bone pathology. The sac is freed down to the entrance of the canal in its entire circumference and is cut completely across as far down as possible. The patency of the lower end of the sac is now determined and if closed it must be incised and opened with scissors. Two or three silk sutures are then introduced through the mucosa and the wall of the sac about 1 to 2 mm. from the cut edge, the sac is lifted upward and protected with a flat retractor. The lacrimal fossa and the adjacent bony structures are now in view (Fig. 3).

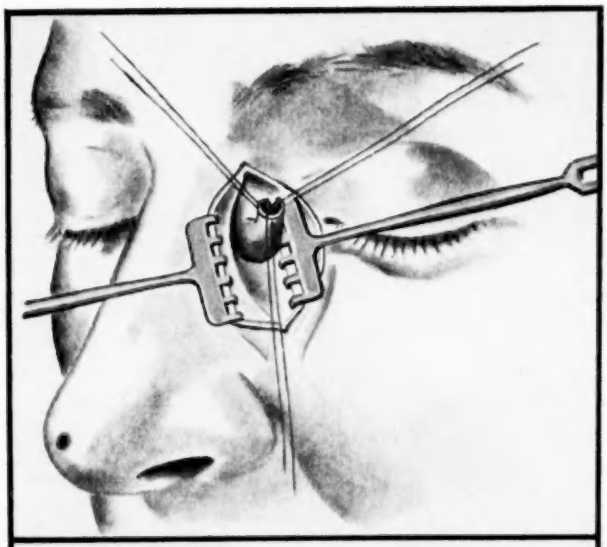

Fig. 3

Perforation of the bone

A trephine 8 to 10 mm. in diameter is applied to the bone toward the lower part of the ascending process of the maxilla and should be directed slightly downward and as nearly as possible at right angles to the plane of the intranasal surface. The bony opening includes part of the anterior lacrimal crest and lacrimal sulcus anterior to where the superior maxilla unites with the lacrimal bone. The window thus made should lie almost entirely in the nasal process of the superior maxilla. Since the bone is much thicker in front than behind, the trephine must penetrate more deeply anteriorly. In order to prevent slipping, a trephine with a center pin must be used. It is well not to drill the hole too far upward or too far backward as air cells may be encountered. The trephine in use is actuated by an electric motor and care must be taken to protect the soft tissues. This is best accomplished by the introduction of lid retractors, which will hold the soft tissues and the sac from the field of the rapidly revolving drill (Fig. 4). After perforation, the bone and corresponding nasal mucous membrane is removed. The nasal mucous

membrane is trimmed flush with the edges of the opening in the bone. The packing is withdrawn from the nose and a spatula is passed through the nostril to insure that the opening is free with no overhanging tags of mucous membrane. A good sized probe is now passed through the old naso-lacrimal duct and the mucous membrane in this canal is destroyed by means of a curette.

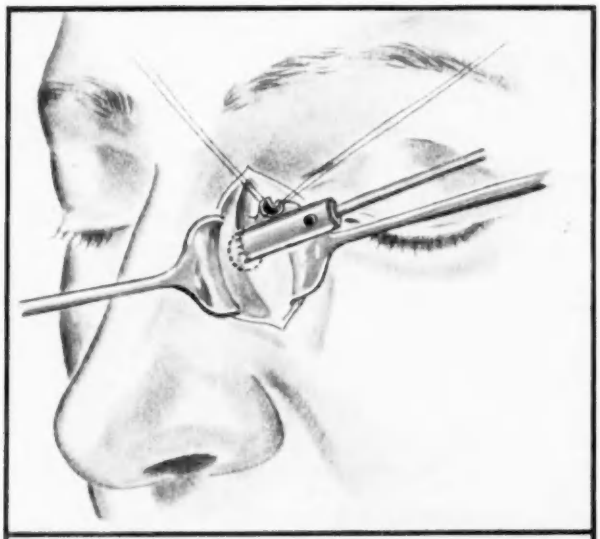

Fig. 4

Implantation of the sac

The ends of the silk sutures which are holding the lower end of the sac are now passed through the new opening into the nose and are picked up with a pair of nasal forceps. This is not difficult, because the new opening is not under the middle turbinate but is slightly anterior to the anterior end of the middle turbinate (Fig. 6). The sutures drawn through the nostril hold the sac in the new bony canal. The sutures are held in place on the side of the cheek with adhesive tape (Fig. 5). Possibly it would be better to tape the sutures on the opposite cheek for more direct traction. A small plug of gauze is inserted into the nostril and may be changed on the second day to avoid odor.

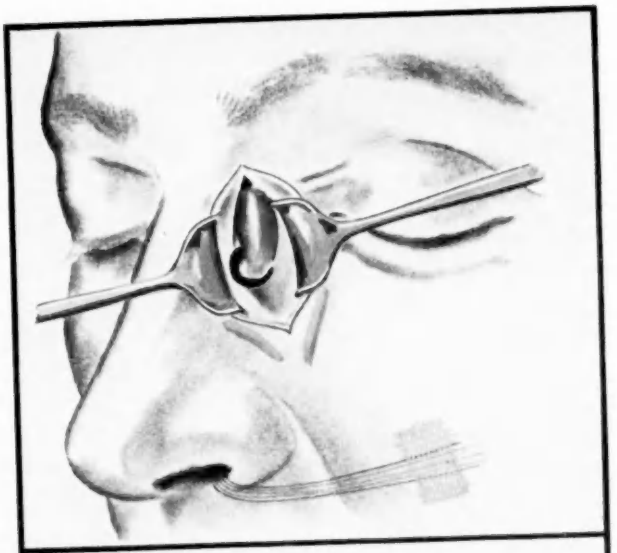

Fig. 5

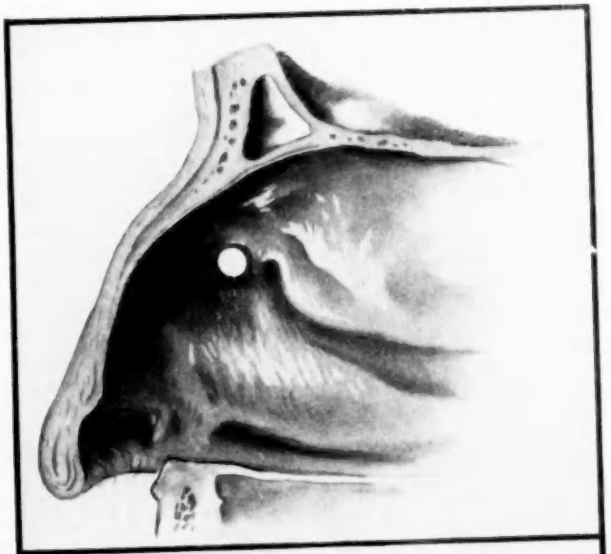

Fig. 6

Skin suture and dressing

An imperceptible scar is obtained if the muscles and fascia are sutured with fine catgut. The skin over it is carefully approximated with horsehair, using a subcuticular type of stitch. Pads of gauze saturated with vaseline are now placed over the incision and a firm pressure bandage is applied.

After-Care

The patient remains in bed for the first day but is allowed up on the second. On the fourth day the skin stitch is removed. On the sixth day the sac sutures are withdrawn and the canaliculus is irrigated with normal saline solution. The pressure bandage is now left off. For a week more the new passage is irrigated daily with normal saline. Probing is not necessary and may actually do harm.

Results

In May, 1935, I[25] made a preliminary report on the transplantation operation and up to the present writing 42 consecutive operations have been performed on 39 patients — 28 females and 11 males. Four of these were children between the ages of 6 to 13 years. The average age, exclusive of children was 48 years. Where the general condition warranted, operations were performed on several patients in the seventh decade. All were unselected cases of chornic dacryocystitis. Although a simple mucocele was present in the great majority of the patients, in 11 there had been one or more previous attacks of acute dacryocystitis and an external fistula was present at the time of the operation in 10 cases. In all patients the obstruction was low down in the bony canal as determined by the introduction of a small probe.

All of the 39 patients complained of epiphora at the time of examination and had visible secretion in the conjunctival cul-de-sac; pus could be expressed by pressure over the sac. These symptoms had been in evidence for a number of years.

Previous treatment, before admission to the eye clinic, had been by repeated probing in 26 patients, some of them over a period of years, and in 7 patients the lower canaliculus had been slit for the purpose apparently, of passing probes.

The period of observation, after the transplantation operation had been performed, was from three months to four years, an average of eighteen months in 39 cases.

The criteria of success were the free passage of fluids into the nose and the absence of all symptoms referable to the lacrimal drainage apparatus. On the follow-up all patients were examined personally except two, who replied by letter.

There were three failures:

1. Case No. 8, a female child, 6 years old. At the time of the operation a small atrophic sac was found, stenosed at its lower end. All symptoms returned one month after operation. The child was re-operated later and the sac was found to be closed. A Toti-Mosher operation performed at this time cured the infection but did not restore drainage into the nose.

2. Case No. 13, female, age 30. Examination of the nose before operation showed a deviation of the septum. The middle turbinate was unusually large, the anterior tip of which was overhanging the area of trepanation. The patient refused nasal surgery as a preliminary step. Trephining of the bone placed the opening underneath the tip of the middle turbinate and adhesions closed the sac completely. I feel that in this case the failure could have been avoided by a submucous resection with removal of the anterior tip of the middle turbinate.

3. Case No. 21, female, age 62. This patient had an acute phlegmonous dacryocystitis which was treated in the usual manner and at the time of the examination had a chronic dacryocystitis with fistula formation. Obstruction was in the bony canal. The nose showed a marked deviation of the septum and the tip of the middle turbinate was overhanging the area of trepanation. This patient also refused preliminary nasal surgery as a corrective measure. The cause of failure was the same as in Case No. 13 and could, perhaps, have been avoided.

Of the 39 patients there were 7 who previous to entering the clinic had their lower canaliculi slit for the purpose of passing probes. In all of these 7 patients there was some visible tearing months after an otherwise successful transplantation operation. An ideal result is therefore not achieved if the canaliculi have been slit. This, however, is not a fault of the transplantation operation.

Data on 39 Cases of Transplantation Operations on the Tear Sac

	HISTORY AND TREATMENT ELSEWHERE											EXAMINATION AND OPERATION						RESULTS AND FOLLOW-UP	
No.	Name	Sex	Age	Eye	Onset Time	Tearing	Secretion Expressed	Swollen Sac	Acute Dacryo.	Fistula	Probing	Operation	Slit Punctum	Position of Obstruction	Nose	Operation Date	Finding at Operation	Tearing	Results	Period of Observation
1	EJH	M	58	L	3-4 yrs.	+	+	+	0	0	+ 2 yrs.	0	0	Bony canal	Good	6- 6-34	Large mucocele	0	Good in 1938	4 years
2	EEB	F	56	L	Many years	+	+	+	0	0	+ 1 yr.	0	0	Bony canal	Good	12-29-34	Large mucocele	0	Good in 1938	3 years and 6 months
3	ME	F	71	R	Many years	+	+	+	0	0	+ 3 yrs.	0	0	Bony canal	Good	1-23-35	Large mucocele	0	Good in July, 1936 Patient died later	1 year and 6 months
4	HEF	M	64	R L	2 yrs.	+ ou	+ ou	+ ou	+ R-3 mos. ago	+ R	2 yrs.	Incised phleg. dacryo. rt 3 months ago	0	Bony canal fistula	Good	OD 2-17-35 OS 2-27-35	Large mucocele ou	0	Good ou. in 1938	3 years
5	GD col.	F	40	R	6 mos.	+	+	+	+ 2 wks ago	+	Svrl. times	Incised phleg. dacryo. 2 weeks ago	0	Bony canal fistula	Good	3-27-35	Fistula	0	Good	3 years
6	LS	F	35	R	Svrl. yrs.	+	+	0	+ 2 mos ago	+	+ many times	Incised phleg. dacryo. 2 months ago	0	Bony canal fistula	Good	8-17-35	Mucocele fistula	Some at first	Good	3 years
7	KC	F	60	L	Svrl. yrs.	+	+	+	0	0	+ svrl times	0	0	Bony canal	Good	9-17-35	Mucocele	0	Good	2 years—died
8	MR	F	6	L	Svrl. yrs.	+	Small amt.	0	0	0	+ many times	0	0	Bony canal	Good	9-25-35	Atrophic Sac Peridacryo.	+	Failure. Did Toti-Mosher op. 7-25-36. Sac obliterated	Failure. Did not irrigate through
9	JM	M	7	R	Svrl. yrs.	+	+	0	+ 1 mo. ago	+	0	Incised phleg. dacryo. 1 month ago	0	Bony canal fistula	Good	10-19-35	Mucocele fistula	0	Good	2 years and 6 months
10	WFP	F	40	L	1 yr.	+	+	+	0	0	+ svrl times	0	0	Bony canal	Good	1-22-36	Mucocele	0	Good	1 year and 4 months (Patient moved away)
11	VB	F	37	L	Svrl. yrs.	+	+	+	0	0	+	0	0	Bony canal	Good	2-13-36	Mucocele	0	Good	2 years and 6 months
12	WR	M	43	R	1 yr.	+	+	+	0	0	0	0	0	Bony canal	Good, a few polyps removed	2-29-36	Mucocele	0	Good	2 years and four mos
13	PP	F	30	R	12 mos.	+	+	+	0	0	0	0	0	Bony canal	Large m.t. Should have ant. 2/3rd removed	3-18-36	Mucocele	+	Failure 5-10-37	Opening under m.t. closed. Refused to have anything further done

Data on 39 Cases of Transplantation Operations on the Tear Sac

	HISTORY AND TREATMENT ELSEWHERE								EXAMINATION AND OPERATION										RESULTS AND FOLLOW-UP	
No.	Name	Sex	Age	Eye	Onset Time	Tearing	Secretion Expressed	Swollen Sac	Acute Dacryo.	Fistula	Probing	Operation	Slit Punctum	Position of Obstruction	Nose	Operation Date	Finding at Operation	Tearing	Results	Period of Observation
14	EH	F	67	L	4 yrs.	+	+	+	0	0	+	0	+ for prob.	Bony canal	Good	5-25-36	Mucocele	+	Good	2 years. Eye st-ill wat ers a little
15	OS	F	63	R	For yrs.	+	+	+	0	0	+ many times	0	0	Bony canal	Good	7-29-36	Mucocele	0	Good	2 years
16	WC	M	45	R	Svrl. yrs.	+	+	+	0	0	0	0	0	Bony canal	Good	7- 6-36	Mucocele	0	Good	2 years
17	SM	F	52	L	3 yrs.	+	+	+	0	0	0	0	0	Bony canal	Good	9- 1-36	Mucocele	0	Good	1 year and 10 months
18	JH	M	13	L	1929	+	+	+	0	0	33x in 2 yrs. No. 9 probe	0	+ for prob	Bony canal	Good	9-13-36	Mucocele	+ slit petm.	Good	1 year and 10 months
19	JK	F	48	R	15 mos.	+	+	looked like tumor	0	0	0	0	0	Bony canal	Good	11- 5-36	Mucocele	0	Good	1 year and 8 months
20	EMC	M	61	R L	3 yrs. ou.	+	+	+	1 yr. ago ou.	R+	+	+ years ago for acute dacryocystitis	0	Bony canal	Good	3-1-37 O.D. 3-8-37 O.S.	Fistula and mucocele	0	Good	6 months—excellent
21	TMR	F	62	R	12 mos.	+	+	0	+ 1 mo. ago	+	0	+ 1 mo. ago for acute dacryocystitis	0	Bony canal	Deviated septum, large m. t.	4-27-37	Small mucocele	+	Failure (in 1 mo.)	Should have had tip of m.t. rem'd. Opening under middle turbinate
22	VW	M	26	L	3 yrs. trauma	+	+	+	0	0	0	0	0	Bony canal	Good	6-16-37	Mucocele	0	Good	1 year
23	PL	F	46	R	Many yrs.	+	+	+	+ 6 mos. ago Uni. H.	+	0	+ 6 mos. ago. Incised acute dacryocystitis	0	Bony canal	Good	7-15-37	Mucocele and fistula	0	Good	1 year
24	IS	F	56	R	10 yrs.	+	+	+	0	0	+ svrl times	0	0	Bony canal	Good	8-26-37	Mucocele	0	Good	1 year
25	WW	F	56	L	Svrl yrs.	+	+	+	0	0	+ many times	0	0	Bony canal	Good	8-27-37	Mucocele	0	Good	1 year
26	RS	M	18	R	1 yr. trauma	+	+	+	0	0	0	0	0	Bony canal	Had submuc. op. ant. tip m.t's rem'd 3 wks. prior to operation	9-10-37	Mucocele	0	Good	10 months

Data on 39 Cases of Transplantation Operations on the Tear Sac

	HISTORY AND TREATMENT ELSEWHERE												EXAMINATION AND OPERATION					RESULTS AND FOLLOW-UP	
No.	Name	Sex	Age	Eye	Onset Time	Tearing	Secretion Expressed	Swollen Sac	Acute Dacryo.	Fistula	Probing	Operation	Slit Punctum	Position of Obstruction	Nose	Operation Date	Finding at Operation	Tearing	Results	Period of Observation
27	LF	F	63	R	1 yr.	+	+	+	+ 2 wks. ago Univ. H	+	+ many times	2 weeks ago incised acute phlegmon daeryocystitis	+	Bony canal	Good	9-17-37	Mucocele and fistula	+ slit petm.	Good	10 months
28	FHW	F	49	L	Many yrs.	+	+	+	+ 5 mos ago	+	+ many times	Incised 5 months ago. Acute phlegmon	0	Bony canal	Good	9-24-37	Mucocele and fistula	0	Good	10 months
29	CA	F	19	R	Since child	+	+	+	0	0	+ once a wk., past 2 yrs	0	0	Bony canal	Good	10- 8-37	Large mucocele	0	Good	10 maths
30	ML	F	24	R	Since child	+	+	+	0	0	+ many times	0	+ for prob	Bony canal	Good	11- 4-37	Large mucocele	+ sli. slit petm.	Good	8 months
31	EEM	F	77	R	Years	+	+ by irrig.	0	0	0	Many times svrl yrs.	0	+ for prob	Bony canal	Good	12- 6-37	Mucocele	+	Good	8 months
32	FC	F	54	R	Years	+	Small amt. upper can trachoma	0	0	0	+	0	+	Lower can closed Bony canal obstruct.	Good	1-26-38	Atrophic sac	+	Irrig. readily into nose through upper canaliculus	7 months
33	MJ	F	61	R	1 yr.	+	+	+	0	0	+	0	0	Bony canal	Good	2- 4-38	Mucocele	0	Good	6 months
34	EB	M	10	R L	8 yrs. trauma	+	+	+	0	0	Many times	0	0	Bony canal	Good	O.D. 3-9-38 O.S. 3-12-38	Large mucocele ou.	0	Good	5 months
35	DR	F	49	L	Svrl. yrs.	+	Through upper canal	0	0	0	Many times	0	Ir + can ob's't	Bony canal	Good	3- 9-38	Mucocele	+	Good through upper punctum	5 months
36	DS	F	20	L	4 yrs.	+	+	0	+ 4 yrs. ago, recur. 5x	+ 4 yrs	0	0	0	Bony canal fistula	Good	4- 4-38	Mucocele and fistula	0	Good—external fistula closed 1 mo. later with cautery	4 months
37	AB	M	81	R	2-3 yrs.	+	Very little	0	0	0	+ svrl.	0	0	Bony canal	Good	5-11-38	Small mucocele	0	Good	3 months
38	EJ	F	44	L	1 yr.	+	+	+	0	0	0	0	0	Bony canal obstruct	Good	6-21-38	Mucocele	0	Good	3 months
39	EN	F	31	L	Svrl. yrs.	+	+	+	+ svrl. times	0	+	Acute dacryo. Incised twice a year since 1936	0	Bony canal	Good	7-14-38	Mucocele	0	Good	2 months

CONCLUSIONS

1. In reporting this procedure an attempt has been made to take advantage of the best features of the previously reported techniques of the transplantation operation, and a method has been developed which it is believed will reduce the failures to a minimum.

2. Co-existing nasal pathology must be corrected as a preliminary step in the operation.

3 Chronic cases with large sacs from which a sizeable quantity of pus can be expressed on pressure are best suited for this operation.

4. Chronic dacryocystitis with fistula formation is not a contraindication to this operation. The fistula closes in most instances as soon as drainage into the nose is re-established.

5. None of the sac is sacrificed and no cul-de-sac is left wherein infectious material may collect. Natural drainage clears up the infection in the sac.

6. Less bone is removed and there are no flaps of mucous membrane to deal with. There is practically no reaction in the nose.

7. Not much more time is required for this operation than for a simple excision of the sac. It is the least difficult of all dacryocystorhinostomy operations.

8. Little or no after-treatment is required and the *scar* is imperceptible.

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DISCUSSION

DR. RALPH O. RYCHENER, Memphis, Tenn.: I concur heartily with the essayist in finding no place for extirpation of the lacrimal sac save in malignancies of that area. Epiphora persists after such an operation and although there may be some sections of the country in which this symptom does not occur (I was told in Richmond that it was a negligible complaint

in Virginia), I have not found it so in Michigan or Tennessee. Dacryorhinostomy is logical even in cases in which one desires to do intra-ocular surgery at a later date, for the re-establishment of normal function permits drainage and the discharge of infected material into the nose. Chilow found that pneumococci disappeared from the conjunctival sac after dacryorhinostomy simultaneously with the disappearance of the discharge of mucopus from the sac. Cirincione proved that pathogenic organisms would not readily travel up the lacrimal passage after dacryorhinostomy by packing tampons soaked in bouillon cultures of bacteria against the new opening in the nose. After twenty-four hours cultures from the conjunctival sac were negative. However, West and Boumke found pneumococci persisting on the conjunctiva in forty-three per cent of cases following extirpation of the sac.

While mentioning tumors of the sac it may be well to call our attention to the fact that mucocele of the sac may sometimes simulate tumor. The sac becomes blocked at the internal punctum as well as in the bony canal below and the contents of the sac undergoing degeneration cause the sac to bulge, becoming round and firm, withstanding indentation. I have seen several cases diagnosed as tumors of the sac, in which, after the application of heat and considerable pressure, the contents of the sacs were regurgitated upon the conjunctiva, and I am sure others have had a similar experience.

There are five generally accepted methods which are used for restoring physiological action of the lacrimal passageways in the treatment of chronic dacryocystitis, and in reviving the Speciale-Cirincione transplantation operation Dr. Stokes has illustrated the fate of the other four which have likewise been advocated, discarded and then again revived on the strength of reports of modifications with successful results by later authors. The absence of unanimity of opinion as to the best technique means that most operators have encountered failures in their preferred procedures and have been induced to try other techniqus for establishing patency of the lacrimal passageway.

The other four procedures are the external operation of Ohm popularized by Dupuys-Dutemps and Bourguet; the internal operation of Caldwell, effectively modified by West and Halle; the combined technique of Toti-Mosher; and finally the methods grouped under the heading of "giant probings" in which an opening is forced into the nose and a metal stylet is left in place for varying periods of time.

For the past three years I have been familair with the operative technique described by the essayist. Quite satisfactory as it is in his hands it does not seem to me to be a procedure which is suitable for every case of chronic dacryocystitis. By his own operative results he has established that it is ineffective in the case of the small atrophic sac. It will not suffice in sacs which are obstructed by papillomata nor will it be satisfactory in lacrimal obstructions following trauma of the face in which the sac area is involved in much scar tissue. One can, of course, outline the sac by the injection of a radio-opaque substance and by X-ray determine whether the sac is suitable for transplantation and if not, some other approach may be used.

Although the experience of my group has not been as large as that of the essayist, I have since 1929 used the Dupuys-Dutemps modification of the external operation with very satisfactory results. My cases have been derived practically entirely from office practice and have included all of the usual findings previously mentioned. My patients have ranged in age from six to seventy-two and were about evenly divided with regard to sex. There have been twenty-seven operations done on twenty-four patients and a successful result has been achieved in every case. It has therefore been difficult for me to bring myself to a trial of the transplantation procedure for one hesitates to change a method which in his hands has been so perfectly satisfactory.

Although statistics show that the external operation ranks as the most successful of all the procedures devised to effect a new lacrimal route to the nose, it too has had its modifications as illustrated by the latest, devised by Paul Chandler who makes three mucous membrane flaps between the sac and nasal mucosa instead of the customary two. Some criticism has been made about the difficulty in suturing the flaps in this particular operation. This difficulty can be entirely eliminated by the use of several simple adjuncts to the ordinary equipment. A good light as from a reflecting mirror or head lamp is essential. The use of a suction tip insures a clean field. The deep insertion of the ordinary lacrimal sac retractor will dispense with one assistant and rid the operative field of several extra instruments. One of the greatest boons has been the development of the atraumatic suture and if this is used with a small, half curved needle and an ordinary small hemostat used as the needle holder rather than all the fancy articles devised for this purpose, one will experience no great difficulty in the insertion of the sutures.

Dr. Frank E. Burch, St. Paul, Minn.: Dr. Stokes' paper again emphasizes conservation of the lachrymal sac versus extirpation and loss of physiologic function in lachrymal drainage. His cases prove the virtue of the method he has employed and illustrated for us, and his paper stresses the importance of anatomical relationship.

It is necessary to study the condition of the nose in each individual case. Schaeffer and Onodi established an anatomical fact which should be well known to every ophthalmologist attempting dacryocystorhinostomy, one also proven by Whitnal, who found the upper half of the lachrymal fossa overlying anterior ethmoid cells in most specimens he examined, the cells often extending completely across the fossa.

In attempting any modification of the Toti operation, especially those advocated by Cirincione, Mosher, Stokes and others, the greatest freedom from underlying ethmoidal cells will be found near the junction of the fossa and duct. In making a new foramen into the nose, therefore, it is especially important that this be made *low down* and *well forward*, cutting away the anterior lachrymal crest through fairly thick bone. Those of us who are not otolaryngologists or rhinologists are at a slight disadvantage in meeting some of the nasal conditions which arise, but by following the procedure recommended and outlined by Dr. Stokes, also emphasized by Dr. Mosher, we only occasionally find it necessary to call in a rhinologist

to correct an unusually tight nasal passage, and remove the middle turbinate. There is no doubt in my mind about the relative value of utilizing the upper end of the duct with the lower portion of the sac for drainage purposes. In those of my own series (dacrocystorhinostomies), which I have been able to follow up, the higher percentage of success has been in patients in whom the thicker anterior portion of the crest has been thoroughly removed, including a goodly portion of frontal process of the superior maxilla and entirely avoiding the upper and posterior half of the lachrymal fossa. Not only must pre-existing nasal pathology be dealt with, but patients should be given to understand that subsequent *cleansing* of a short-circuited sac must be continued for a time; likewise subsequent treatment of nasal infection (usually ethmoiditis) and occasional probing of adhesions may be necessary. Inadequate followup treatment is the cause of some failures.

Dr. Stokes has definitely showed that fistula is no contra-indication to this operation. It was present in 9 of his 39 cases. I was somewhat surprised to note that so many of his cases showed normal noses (only 4% abnormal), and I would like to ask Dr. Gifford if his cases were selected on this account. Two of his failures apparently were due to failure to correct abnormal nasal pathology.

One of the advantages of the procedure advocated and carried out by Dr. Stokes is the freedom from *necessity* of suturing nasal and lachrymal mucosa. Personally, I have sometimes found suturing difficult but facilitated by special instruments. I believe there is distinct advantage gained by dissecting out the head of the naso-lachrymal duct. This was advocated by me before this Academy in 1920. I wish to congratulate Dr. Stokes upon his excellent results with this very practical procedure.

Dr. Harold Gifford, Omaha, Nebr., in closing: I wish to thank Dr. Burch and Dr. Rychener very much for their discussion and I am sure the criticism was justified. Dr. Burch said the cases might have been selected because of absence of nasal pathology, but they were not; it just happened that way. They were consecutive cases taken as they came to the clinic and office.

Two points, one of which I think is very important. In trephining, you do not get any serious hemorrhage after the operation. In the Toti-Mosher operation or any operation that goes back into the lacrimal bone, there is danger of cutting the ethmoid arteries and having serious bleeding. In this operation you get no serious post-operative bleeding. The patients go home the day after the operation with pressure bandage in place.

This operation does not work very well in children. Where the bridge of the nose is flat and the sac is very tiny there is not enough sac to bridge this long distance between the external and internal surfaces of the trephine opening.

Dr. Frank N. Knapp of Duluth sent us his paper, wherein he suggested the skin incision be made only through the skin and then separating the muscle fibers by blunt dissection. This may eliminate some of the bleeding. I think that it is a good point.

OCULO-GLANDULAR DISEASES WITH SPECIAL REFERENCE TO TULAREMIA AND PARINAUD'S CONJUNCTIVITIS

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LONGVIEW, TEXAS

In the last eight years I have had the opportunity to see twenty-three cases of oculo-glandular disease in my practice, and it has been suggested to me that a brief description of the series would have interest, especially since in several of the cases colleagues whose ability in these lines is well known to you have generously helped out my own deficiencies of experience and resources for pathologic and bacteriologic study. Nineteen of these cases were briefly reviewed in a previous report.[1]

In all of the cases of my series there was a more or less generalized conjunctivitis and a vegetative growth springing from the conjunctiva combined with swelling of one or more of the regional lymphatic glands. This association of symptoms was first definitely called to the attention of the medical world by Parinaud in a description of three cases, published in 1889, and it has since gone under his name as Parinaud's conjunctivitis. The oculo-glandular syndrome is now regarded as unusual but not extremely rare. My own experience leads me to believe that it is of more common occurrence than the literature on the subject would lead one to suppose. It is usually a mild disease with spontaneous, though delayed, healing and no sequelae, and it seems probable that many cases run their course without ever having been recognized as such. Other than removal of the large vegetative growths, treatment does not seem to alter the course of the disease.

The clinical picture described by Parinaud is clear-cut and it is remarkable that in half a century it has been little added to or otherwise altered. For a description in words one cannot do better than to turn to Parinaud himself. Somewhat shortened, his description is as follows:[2] "The conjunctiva is the seat of red or yellow vegetations, semitransparent at the beginning, opaque at a

later stage, which can reach the size of a large pin-head. Alongside of these fleshy granulations, one finds very minute ones, entirely yellow. The cornea does not appear to have any tendency to be involved. There is a mucous secretion. The lids are swollen, firm to the touch. The parotid region becomes very rapidly the seat of an inflammatory swelling which can extend to the neck and in the midst of which one discovers swollen and sometimes softened glands. There is fever with irregular chills; it can persist for a long time, but always remains moderate and does not gravely react on the general state."

Very rarely the condition is bilateral. Hoor[3] reported two such cases. My series contains one. From the history given in my case it does not appear that there was any noticeable interval between the development of the conjunctivitis in one eye and its appearance in the other. The preauricular gland was enlarged on each side.

In general, the glandular swelling is more or less simultaneous with the occurrence of the conjunctivitis. In one of my cases, however, the enlargement of the glands appeared more than two weeks after the first eye symptoms were noticed, and in another case the glandular swelling was present six weeks or longer before the eye became reddened. The glands are often tender, distinctly painful, and sometimes they suppurate.

Constitutional symptoms were absent or slight in the great majority of cases in my series. Onset was mild and the symptoms developed gradually. Two cases, on the other hand, are singled out by a clinical course that was strikingly different from that of all the others. Onset was rapid, with severe constitutional symptoms and very high temperature, reaching 106° F. in one case and 105.6° F. in the other. These two cases, furthermore, were the only ones of the series in which the lesion of the eye ulcerated. More will be said about these two cases when I come to speak of etiology.

Interest in the oculo-glandular syndrome centers today in the problem of etiology. Parinaud made no pathologic or bacteriologic studies. He pointed out that the cases which he was reporting gave the clinical impression of an infectious disease, and he suggested that the disease might be contracted from animals.

That the disease was a manifestation of tuberculosis was early suggested. In a few cases the tubercle bacillus has been demonstrated in material from patients' lesions, and animal inoculation with such material has produced lesions similar to those of tuber-

culosis. von Szily[4] recently demonstrated tuberculosis after painstaking study in a case that was, clinically, typical Parinaud's conjunctivitis. He believes that the infection was of the bovine variety. Nichelatti[5] thinks that Parinaud's conjunctivitis results from tuberculous re-infection of an allergic individual, which would explain the difficulty in finding the bacilli in the lesions and the spontaneous healing.

On the other hand, it has been shown that the oculo-glandular syndrome can be caused by Bacterium tularense, by either of two organisms which are probably related to this bacterium — Bacillus pseudo-tuberculosis rodentium and an organism named by Paschcff,[6] its discoverer, Micrococcus bacillus polymorphicus necroticans — and by the leptothrix, first isolated from cases of the oculoglandular syndrome by Verhoeff, and since found in large series of cases by Sanford Gifford and others.

Analyzing my twenty-three cases from the standpoint of etiology, I am able to state that in two cases the patients' blood agglutinated with Bacterium tularense, in four cases the leptothrix was discovered, but in the remaining seventeen cases no causative organism was demonstrated in spite of the fact that in four of these, biopsies were made of the conjunctival tissue, and several of our leading eye pathologists gave me their invaluable aid in the search. In four of these seventeen cases of undetermined etiology the agglutination test for tularemia was made and was found negative.

Tularemia has recently excited much interest among ophthalmologists both in this country, where it is better known, and abroad, where tularemia in any form has made a recognized appearance only within the last few years. The first case of tularemia in man, which was diagnosed and proved by laboratory methods, was, as related by Vail,[7] a case of tularemia of the conjunctiva. According to Francis,[8] of the United States Public Health Service, 8,137 cases of human tularemia had been reported in this country up to and including 1937. Cases have been reported from every State of the Union, except Vermont and Connecticut, and from the District of Columbia.

The conjunctiva was the primary seat of the infection in 68 or about 1 per cent of the 6,274 cases of tularemia reported in this country up to 1936. Francis[8] states that the infection was transferred to the eye by the hands while dressing rabbits in 52 of the 68 cases.

Certain European authors[9] are today pressing the viewpoint that Parinaud's conjunctivitis and oculo-glandular tularemia are one and the same disease. This view challenges special attention in America, where tularemia in its various forms is so much more prevalent than anywhere else in the world and Parinaud's conjunctivitis is seen at least as frequently as elsewhere. My own experience, which has allowed me to obtain first hand impressions from two cases of proven oculo-glandular tularemia during the time that I was seeing a comparatively large number of cases that were typical of the disease described by Parinaud, leads me to the conviction that oculo-glandular tularemia should be separated entirely from Parinaud's conjunctivitis. To make my reasons for this opinion clear, I shall describe in some detail the clinical picture of my two cases of proven oculo-glandular tularemia.

FIG. 1 — (Hurst) — Lymphadenopathy, seven weeks after primary infection, in case of ocular tularemia.

FIG. 2 — (Hurst) — Conjunctival granuloma with preauricular adenopathy.

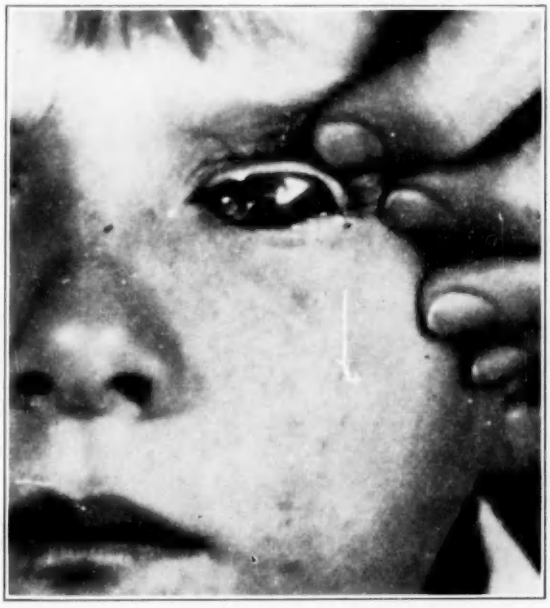

FIG. 3 — (Hurst) — Conjunctival granuloma with preauricular adenopathy.

FIG. 4 — (Hurst) — Lesion on lid in which the leptothrix was found.

CASE HISTORIES OF OCULAR-GLANDULAR TULAREMIA

Case 1. — P. T., a white boy aged 12, entered the hospital January 25th, 1930, because of pain, swelling and redness of the left eye and tenderness, with swelling of the left side of the neck of two days' duration, high fever (temperature up to 106° F.) and general malaise. The patient attributed the swollen eye to a blow from the elbow of another boy, received about eight days previously.

Physical findings: The patient appeared acutely ill. Temperature 104° F. The preauricular, cervical, and submaxillary glands on the left side were enlarged and extremely hard and tender. The throat was generally red, with several small white patches.

Ocular findings: Vision seemed to be approximately normal, but the patient was too ill to permit accurate testing. The right eye appeared normal. The lids of the left eye were markedly swollen and reddened. On the congested palpebral conjunctiva there were many small yellowish ulcers. The other structures of the eye appeared normal.

Diagnosis: A tentative diagnosis of Parinuad's conjunctivitis was made, but in view of the high fever and the seriously ill condition of the patient later in the day, the possibility of tularemia was considered, and questioning brought out the fact that the boy had killed and skinned two wild rabbits one week

before the onset of symptoms. Blood taken five days after the patient entered hospital was reported by Dr. Edward Francis, of the United States Public Health Service, as having positive agglutination for Bacterium tularense in dilutions of 1:28. Blood taken ten days later showed positive agglutination for Bacterium tularense in dilutions of 1:5120.

Clinical course: The patient continued to have fever up to 104° F. for thirty days. Most of the glands on the left side of the face began suppurating after about one month and discharged for several weeks. The conjunctiva returned to normal at the end of six weeks.

Case 20. — B. M. Negro male, aged 21, was first seen in consultation September 4th, 1937. He had been injured in the right eye one week previously by being struck by the branch of a tree. Three days after the injury fever appeared and the temperature rose to 104° F. The injured eye was reddened, swollen, and painful, there was pain in the head and shoulders, sore throat, and general malaise. The patient had been confined to the hospital, where a thorough general examination had revealed no cause for the fever other than what appeared to be a mild tonsillitis.

Ocular findings: Examination of the right eye showed a deep corneal abrasion in the pupillary area, with some swelling of the lids and more than usual injection of the palpebral and bulbar conjunctivae. There was very little secretion in the conjunctival sac. The right eye was otherwise normal. The left eye appeared entirely normal. Since the patient was acutely ill, his vision was not tested.

Diagnosis: A clinical diagnosis of tonsillitis and traumatic injury to the left eye was made at this time. The diagnosis of tularemia was not entertained until fourteen days later. The patient's blood was then tested for agglutination with Bacterium tularense and was found positive in dilutions of 1:640.

Clinical course: After the time of my first examination, the patient continued to run a very high fever, and in about seven days the right preauricular and submaxillary glands enlarged. Fourteen days after the first consultation, he was seen again by me. He had been seriously ill, with temperature up to 105.6° F., and no diagnosis had been made, though he had been observed carefully by his physicians and had had thorough laboratory checks. Examination of the right eye now showed typical lesions of tularemia

of the palpebral conjunctiva, with nodular ulcerations having yellowish centers, the picture so vividly described by Dr. D. T. Vail, Sr.[10] as looking "like yellow polka dots in a piece of turkey red calico." The corneal wound had not healed, as would have been expected, but remained as an excavated ulcer. The preauricular and submaxillary glands were greatly swollen and very tender. At this time the diagnosis of tularemia was established by agglutination test. The patient continued to run a high fever for six weeks after the onset of the disease. The lymphatic glands did not suppurate. When the patient was last seen, four months after onset of the disease, the conjunctiva had returned to normal, but a large scar was present in the cornea, which reduced the vision to light projection.

The occurrence of trauma to the eye at about the time of the contraction of the infection in both my cases of tularemia may be worthy of note. Francis[8] states that since the organism will penetrate normal skin, a wound of entry is not necessary for infection, but that in the majority of human cases a wound of entry has been inflicted at the site of cutaneous infection either at the time of infection or shortly after or before.

For comparison with these cases of proven oculo-glandular tularemia, I will describe more briefly the clinical features of a case in which Dr. Sanford Gifford demonstrated the leptothrix in sections of the conjunctival vegetation.

CASE HISTORY OF LEPTOTHRICOSIS

Case 21. — P. N., a white girl, aged 8, first noticed a swelling in front of the left ear one week prior to entering hospital. The swelling was hard and slightly tender. Two days before admission the left eye was swollen. There were no other symptoms on admission. Examination showed a hard, non-tender, fixed left preauricular gland measuring about 3x3x3 mm. The upper lid of the left eye was diffusely reddened, there were numerous follicles, and a polypoid vegetation, measuring 1x2x3 mm., springing from the center of the upper fornix. The temperature was 98° F., rising to 101° F. later that day. During the following six days the temperature oscillated between 97° and 100° F. Recovery was complete in three weeks.

The following case is described as an example of a case of the oculo-glandular syndrome with unknown etiology. The histologic sections were examined by Verhoeff and failed to disclose the leptothrix or to provide the basis for any other etiologic diagnosis.

CASE HISTORY OF OCULAR-GLANDULAR SYNDROME OF UNKNOWN ETIOLOGY

Case 4. — C. E. S., a white boy, aged 14, had noticed swelling of the lids of the right eye and roughness in this eye for two weeks, but there had been little discomfort and the condition had not interfered greatly with his school work. Examination showed much swelling of the lids of the right eye and on the upper palpebral conjunctiva a growth, the size of a pea, surrounded by enlarged follicles. During the next few days the preauricular and anterior cervical lymph nodes enlarged and became sensitive to pressure. The temperature was 101° F. The blood agglutination test for tularemia was negative. At the end of six weeks the temperature had returned to normal and the conjunctival lesion and adenitis had subsided.

No clinical differences are apparent between those cases of oculo-glandular disease in which the leptothrix appears as the etiologic agent and those cases in which diligent search has failed to disclose the etiology. Nevertheless, I feel that progress will be faster in arriving at the true nature of disease presenting the oculo-glandular syndrome if we hold steadily to an etiologic classification insofar as we can assure ourselves of the etiology in the individual case. Where the tubercle bacillus has been shown to be the etiologic agent, I would term the disease tuberculosis; where the patient's blood agglutinates with Bacterium tularense, I would call it tularemia. Where Verhoeff's leptothrix is found, the logical thing would seem to be to call the disease leptothricosis. It may well be that the future will show the leptothrix in an ever increasing number of cases of typical Parinaud's conjunctivitis, so that we shall be justified in reserving the latter designation exclusively for cases of this etiology. Those cases in which no etiologic agent can be discovered I would place in a group by themselves, as cases of the oculo-glandular syndrome of unknown cause. That there is an organism, or organisms, as yet unsuspected capable of producing the oculo-glandular syndrome with a clinical picture that is typical of Parinaud's conjunctivitis, seems, in consideration of my series of cases, to be strongly probable.

SUMMARY

In twenty-three cases of oculo-glandular disease, with histologic studies on twelve, no clinical difference was noted between cases in which the leptothrix was found and cases in which it

TABLE 1 — *Synopsis of Twenty-three Cases of Oculo-Glandular Disease*

Case No.	*Age*	*Sex*	*Nat.*	*Temp.*	*Glands*	*Etiology*	*Remarks*
1	12	M	W	106.0	Preaur. submax.	Tularemia	Positive agglutination. All glands suppurated.
2	6	F	W	98.4	Preaur. cerv.	Unknown	
3	35	F	W	100.0	Preauricular	Unknown	
4	14	M	W	101.0	Preaur. bilat.	Unknown	Verhoeff reports not typical of Parinaud's. Appearance of rhinoscleroma. Unable to make a diagnosis.
5	23	F	W	101.4	Preaur. cerv. submax.	Unknown	
6	20	F	W	98.4	Preauricular	Unknown	
7	20	F	W	98.0	Preauricular	Unknown	Friedenwald reports unable to make a diagnosis. Does not appear to be T.B.C. or Parinaud's.
8	5	M	W	101.0	Preauricular	Unknown	
9	7	M	W	98.4	Submax. cerv.	Unknown	
10	24	M	W	99.0	Preaur. cerv.	Unknown	
11	13	F	W	100.5	Preaur. bilat.	Unknown	Both eyes involved.
12	15	M	C	99.2	Preaur. cerv.	Unknown	Preauricular glands suppurated and drained several weeks. Cultures from discharge, negative.
13	5	M	W	98.4	Submax.	Unknown	
14	10	M	W	100.0	Preauricular	Unknown	Blood count showed no eosinophiles.
15	60	F	W	99.0	Preaur. submax.	Unknown	
16	29	M	C	98.2	Preauricular	Unknown	Friedenwald reports not Parinaud's. Col. Ash suspects it was. Eos. 3%.
17	26	F	W	100.0	Preauricular	Unknown	
18	33	M	W	98.0	Preauricular	Unknown	
19	9	F	W	99.6	Preauricular	Leptothrix	Leptothrix found by Dr. Lamb. Eos. 3%.
20	21	M	C	105.6	Preaur. submax.	Tularemia	Agglutination positive. No suppuration of glands.
21	8	F	W	98.0	Preauricular	Leptothrix	Leptothrix found by Dr. Gifford. Eos. 9%.
22	7	F	W	104.0	Preauricular	Leptothrix	Leptothrix found by Dr. Gifford. Eos. 4%.
23	6	M	W	100.2	Preauricular	Leptothrix	Leptothrix found by Dr. Verhoeff. Eos. 8%.

could not be demonstrated. Two cases of oculo-glandular tularemia were sharply differentiated from the cases of Parinaud's conjunctivitis by their clinical course and agglutination reaction which bears out the contention that these are separate disease entities.

I should like to express my appreciation to Dr. F. H. Verhoeff, Dr. Sanford Gifford, Dr. Harvey Lamb of Washington University, Dr. Percy Friedenwald of Johns Hopkins, Colonel Ash of the Army Medical Museum, Dr. J. L. Goforth, and the Department of Pathology of Baylor University, for the bacteriologic and the pathologic study of these cases.

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DISCUSSION

Dr. Sanford R. Gifford, Chicago: Dr. Hurst has had a remarkable experience in being able to see and diagnose twenty-one cases of Parinaud's conjunctivitis and two of oculo-glandular tularemia. Only one other man, Dr. Verhoeff, has had a greater experience with the disease and of the forty-five cases from which he examined material I do not know how many he saw personally. It is remarkable that such a profusion of cases should have been seen in two localities so distant geographically and in every other way, as Boston and Longview, Texas. The only conclusion to be drawn from this is that the condition is more common than is usually believed over a widespread area, and that groups of reported cases accumulate in localities which possess an ophthalmologist interested in the condition. About eight years ago I saw one of Dr. Hurst's cases while at a

meeting in Dallas and could confirm his diagnosis of Parinaud's conjunctivitis. I examined material from this case and found what I considered elements of leptothrix in sections. They were few in number, however, and Dr. Verhoff did not agree with my identification. In material from another case which he saw not long after this, I was unable to find threads or other organisms. In two other cases from which he sent me material this summer, however, many undoubted leptothrix threads were present, in the capillary spaces and free in the tissues. There can be no doubt, therefore that in these two cases at least he was dealing with Leptothricosis conjunctivae.

In eighteen of his cases findings were negative. As I understand it, these cases were clinically similar to the ones in which Leptothrix was found. Dr. Hurst states that in 12 of these cases tissue was not removed for sections and it seems more than possible that leptothrix might have been found in some of these. I would like to emphasize the simplicity with which such material can be taken. Nothing need be said to the patient. Two or three applications of 10% cocaine are made to the conjunctiva at the site of a lesion and after waiting three minutes it is removed by one snip of a very sharp iris scissors. In one of Dr. Hurst's cases and in several of mine, where one especially large granulation was present, such removal has seemed to hasten clearing up of the lesions definitely. Dr. Hurst has not mentioned that in some cases the granulations are very small or even absent, but that there are present in all cases small grey areas which Verhoeff has described as points of focal necrosis. These seem to be the essential lesions, and it is in these areas that leptothrix threads are found. In at least one of Dr. Hurst's cases in which I found many leptothrix threads, many giant-cells were present, a finding which, I believe, differs from those of Dr. Verhoeff. Aside from the four cases from which I saw only the material, I have seen ten cases of the condition in Nebraska, Illinois, and Texas. In three of these, the first cases I had seen, sections were negative. In one permission to remove material was refused. In six cases sections showed the elements of a thread-mold which, in view of the positive cultures of Verhoeff and King, one must be disposed to accept as a variety of Leptothrix (slides). In one case the material was from the only lesion present at the time it was removed and portions of a vegetable foreign body were found embedded in the tissue. Hence in this case, it seemed that the primary lesion was removed, and that inoculation had occurred by the embedding of this vegetable particle in the upper cul de sac. These cases have been reported elsewhere, except for two seen this year. One of these was in a patient with unilateral adenopathy and one small polypoid mass in the upper fold. Many elements of a thread-mold were found in sections (see slide) and cultures according to Verhoeff's method made by Dr. Day from the pre-auricular gland showed a leptothrix. The case will be reported in more detail elsewhere. Cultures from the gland in another case were not made until most of the swelling had disappeared, about six weeks after the onset, and were negative. Since the report of Verhoeff and King, Wright has grown leptothrix from the pre-auricular gland of a case.

It is unfortunate that demonstration of the leptothrix in sections and its growth in culture should both be slightly complicated procedures. Cul-

tures require that special media and facilities for partial carbon dioxide tension be prepared beforehand and demonstration in sections requires removal of one of the essential lesions, its fixation in Zenker's fluid and some experience with Verhoeff's modified Gram stain to prevent complete decolorization of the threads. It is undoubtedly these details which have prevented identification of the threads by a larger number of observers.

This has allowed the importance of Verhoeff's discovery to escape the attention of many ophthalmologists, especially in Europe. I became engaged in a mild polemic with Professor Junius of Bonn following his publication in 1932 (Zeit. f. Aug. 1932:78, p. 142). While he was familiar with the reports of Verhoeff and myself, he apparently believed that most cases of the condition described by Parinaud were really cases of tularemia. This was apparently due to the fact that Leptothrix had never been found by a German ophthalmologist. The same point of view was expressed by Pillat and David in 1937 who emphasized the relative frequency of oculo-glandular tularemia. Professor Pillat was finally convinced of the pathogenic importance of leptothrix after Dr. Verhoeff had sent him sections but it cannot be said that my well-meant intervention accomplished very much towards clearing up confusion as to the name of the disease. It is to be hoped that this remarkable collection of cases by Dr. Hurst will come to the attention of many European colleagues.

It still seems to me confusing and misleading to use the names tularemia and Parinaud's conjunctivitis interchangeably. Tularemia is clinically distinguishable from other diseases by the agglutination test which is always positive in the second or third week and usually by a history of contact with rabbits or squirrels. The systemic symptoms are much more marked than have generally been observed in cases described as Parinaud's conjunctivitis. No fatalities have been reported in the latter condition and systemic symptoms are usually limited to slight fever and malaise for a few days after the onset. Agglutination tests for tularemia were made in three of my cases and in two cases seen by Dr. Dillon which were mentioned in a previous paper. They were negative in all as they were in four of Dr. Hurst's cases. During the time when these cases classed as Parinaud's conjunctivitis were seen, I have seen only one case of oculoglandular tularemia. The patient gave markedly positive agglutination tests, was severely ill, and had butchered wild rabbits. This picture agreed with the marked general symptoms seen in Dr. Hurst's two cases of oculoglandular tularemia and those of most reported cases. I have never seen corneal ulceration in Leptothricosis, but it has been reported in cases of tularemia, as in Dr. Hurst's case. This experience gives me a personal impression of the relative frequency of Leptothricosis conjunctivae and tularemia, at least in the middle west of North America. I have seen one case of conjunctival tuberculosis during the same time with positive bacteriologic findings.

The presence of eosinophilia, which was noted in a number of cases of Leptothricosis, is not constant enough to be of positive diagnostic value. It was present in about half of my cases in which differential blood counts were made, amounting to 3.5%, 10%, 4.5%, 8.5% and 8% in five cases of which records are at hand. Dr. Hurst found it in only half of

his cases. Hence it may be considered suggestive but by no means essential to the diagnosis. I believe it has not been reported in tularemia.

It is probable that ophthalmologists will continue to associate the name of Parinaud with the picture of unilateral adenopathy and conjunctival granulations. This is likely to be true in spite of modern refinements which make possible a division of this picture into at least three etiologically distinct entities. Hence the suggestion which I proposed in 1934 still seems a reasonable one, that the general picture be denoted as the *Conjunctivo-glandular Syndrome of Parinuad* and that modern means be employed to differentiate the three diseases which may, in some cases, somewhat resemble each other. These three conditions are, of course, Leptothricosis conjunctivae, the oculo-glandular form of tularemia, and conjunctival tuberculosis. Every case falling in this general syndrome should have an agglutination test for tularemia, examination of excised tissue fixed in Zenker's fluid and stained by Verhoeff's method, examination of excised or curetted material for acid-fast bacilli with guinea pig inoculation when possible, and careful inquiry regarding exposure to wild rabbits or squirrels. Although requiring more preparation, there should be added as exceedingly desirable cultures of aspirated pus on serum agar in containers subjected to 10% carbon dioxide tension.

Dr. Albert C. Snell, Rochester, N. Y.: I just want to call attention to one thing. In Dr. Gifford's remarks, he spoke about taking a piece of tissue without the patient's knowing about it. That is absolutely illegal under common law, and practically every other law in the states of the United States. If you get by with it, it is all right, but you lay yourself open to malpractice suits. Yon are not permitted under the law to remove any tissue from your patient, either dead or alive, without the patient's consent or the consent of those who may have authority over the body.

I thought that might be a warning to some of the members of the society. I think you can always get a specimen if you try hard enough.

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F. REFRACTION

Sidney L. Olsho, M.D., Philadelphia

A continuous course designed to present refraction as a field of inquiry — of interest to those wishing to refresh and inform themselves in the various methods of refraction, writing of prescriptions, etc.

G. PLASTIC SURGERY

This course, originally planned to have been presented by Dr. John M. Wheeler was, because of his death, given by:

Ramon Castroviejo, M.D., New York
Daniel B. Kirby, M.D., New York
Wendell L. Hughes, M.D., Hempstead, N. Y.

H. ELEMENTARY COURSE IN SLITLAMP MICROSCOPY

Robert J. Masters, M.D., Indianapolis
Everett L. Goar, M.D., Houston
Alfred Cowan, M.D., Philadelphia

A fundamental course designed for those wishing instruction in the clinical use of the slitlamp.

I. ADVANCED COURSE IN SLITLAMP MICROSCOPY

1. *Senile Changes as Seen with the Slitlamp*

2. *Senile Cataract*

Robert von der Heydt, M.D., Chicago

Should be preceded by a fundamental course. These two lectures, with slides and photographs, intended for those interested in study with the slitlamp.

J. DISTURBANCES OF OCULAR MOTILITY

Congenital Anomalies

JAMES W. WHITE, M.D., New York

Surgical Treatment of Heterotropia and Heterophoria

JOHN H. DUNNINGTON, M.D., New York

Comparison and Purpose of Various Muscle Tests

LUTHER C. PETER, M.D., Philadelphia

Diagnosis of Heterophoria and Heterotropia

RUDOLPH AEBLI, M.D., New York

Treatment Other Than by Surgery

Use of Orthoptic Instruments

LEGRAND H. HARDY, M.D., New York

A course designed for those desirous of obtaining a complete outline of muscle problems and the solutions.

K. EYE BACTERIOLOGY

PHILLIPS THYGESON, M.D., New York
JAMES H. ALLEN, M.D., Iowa City

A lecture and demonstration (microscopic) course. This course presented the new methods of study as well as the usual routine and accepted methods.

* * *

SINGLE LECTURE COURSES

Ophthalmology

FRANCIS HEED ADLER, M.D., Philadelphia
Physiology of the Ocular Muscles

THOMAS D. ALLEN, M.D., Chicago
Short Review of Uveitis — Malignant; Chronic

S. JUDD BEACH, M.D., Portland, Maine
Refinements of Refraction

CONRAD BERENS, M.D., New York
Focal Infection and Eye Lesions

G. M. BRUCE, M.D., New York
Contact Glasses

FRANK E. BURCH, M.D., St. Paul
Complications of Cataract Surgery

FRANK D. CARROLL, M.D., New York
The Toxic Amblyopias

VIRGIL G. CASTEN, M.D., Boston
Neuro-Ophthalmology

RAMON CASTROVIEJO, M.D. New York
Gonioscopy and Goniophotography

C. P. CLARK, M.D., Indianapolis
Preparation of the Senile Patient for the Removal of Cataract

GRADY E. CLAY, M.D., Atlanta
Rare Lesions of the Fundus Oculi

MARTIN COHEN, M.D., New York
Diseases of the Choroid

FREDERICK C. CORDES, M.D., San Francisco
Anomalies — Congenital and Acquired — of the Optic Discs

MISS SYBIL COX, Cleveland (by invitation)
Reading Difficulties, Etc.

WILLIAM H. CRISP, M.D., Denver
Problems in Refraction — The Use of the Cross Cylinder

ARTHUR M. CULLER, M.D., Dayton
Vaccine and Fever Therapy

M. DAVIDSON, M.D., New York
Occupational Keratitis

E. C. ELLETT, M.D., Memphis
The Choice of Operation for Cataract

JOHN N. EVANS, M.D., Brooklyn
The Interpretation of Central Field Changes

EARLE B. FOWLER M.D., Chicago
Endocrine Disturbances of the Eyes

L. G. HOFFMAN, M.D., Chicago
The Diagnosis and Treatment of Lid Conditions

WENDELL L. HUGHES, M.D., Hempstead, L. I.
Aniseikonia

EDWARD JACKSON, M.D., Denver
Office Practice of Ophthalmology

DEWEY KATZ, M.D., Hartford, Conn.
Histology of the Eye

JOHN E. L. KEYES, M.D., Cleveland
Clinical and Experimental Retinitis

ARNOLD KNAPP, M.D., New York
Diagnosis of Simple Glaucoma with Indications for Treatment

WALTER B. LANCASTER, M.D., Boston
Magnet Operation

LOUIS LEHRFELD, M.D., Philadelphia (by invitation)
Ocular Allergy

ALBERT N. LEMOINE, M.D., Kansas City, Mo.
Ocular Tuberculosis — Diagnosis and Treatment

WALTER I. LILLIE, M.D., Philadelphia
Eye Changes Due to Intracranial Pathology

RALPH I. LLOYD, M.D., Brooklyn
Non-Suppurative Diseases of the Cornea

DONALD J. LYLE, M.D., Cincinnati
Embryological Arrests and Their Effect on Vision

SAMUEL J. MEYER, M.D., Chicago
The Present Status of Retinal Detachment Surgery — Choice of Procedures

AVERY DEH. PRANGEN, M.D., Rochester, Minn.
Myopia: Diagnosis and Treatment

BENJAMIN RONES, M.D., Washington, D. C.
Corneal Dystrophies

C. W. RUTHERFORD, M.D., Indianapolis
Iridocyclitis

R. O. RYCHENER, M.D., Memphis
Affections of the Lacrimal Apparatus

MARK J. SCHOENBERG, M.D., New York
Acute Primary Glaucoma—Diagnosis, Medical and Surgical Treatment

EDMUND B. SPAETH, M.D., Philadelphia
Lacrimal Sac Surgery

SCOTT STERLING, Rochester (Bausch & Lomb Company) (by invitation)
The Types and Mechanism of Bifocals

GEORGE H. STINE, M.D., Colorado Springs
Localization and Diagnosis of Retinal Tears

DERRICK VAIL, M.D., Cincinnati
Optochiasmatic Arachnoiditis

ARTHUR M. YUDKIN, M.D., New Haven, Conn.
Retrobulbar Neuritis

* * *

Otolaryngology

JAMES A. BABBITT, M.D., Philadelphia
Endaural Surgery in Middle Ear and Mastoid Infection

LOUIS J. BIRSNER, M.D., St. Louis
The Surgical Anatomy of the Paranasal Sinuses

L. R. BOIES, M.D., Minneapolis
Surgery of Acute Mastoiditis

C. C. BUNCH, M.D., St. Louis (by invitation)
The Functional Tests of Hearing

LOUIS H. CLERF, M.D., Philadelphia
Differential Diagnosis of Hoarseness

JAMES B. COSTEN, M.D., St. Louis
The Mandibular Joint Syndrome; Anatomy, Pathology, Diagnosis and Treatment

BERT E. ELLIS, M.D., Indianapolis
The Treatment of Intranasal Tumors

I. FREISNER, M.D.,New York
Intracranial Complications of Ear Disease

H. N. GLICK, M.D., St. Louis
Operative Surgery of the Mastoid, Labyrinth and Petrous Apex

MAX A. GOLDSTEIN, M.D., St. Louis
Speech Education for the Deaf

FRENCH K. HANSEL, M.D., St. Louis
Allergy of the Nose and Paranasal Sinuses

VOSS HARRELL, M.D., Detroit
The Treatment of Mastoiditis with Special Reference to Mastoiditis in Contagion

LYMAN H. HEINE, M.D., Fremont, Nebr.
Examination of the Patient

FREDERICK T. HILL, M.D., Waterville, Maine
Local Anesthesia in Otolaryngology

KARL M. HOUSER, M.D., Philadelphia
Sulfanilamide Therapy in Otolaryngology

ABBOTT T. HUTCHINSON, M.D., New York
The Mastoid with Its Radical Modifications. A Reiteration, with Readjustments Toward Better Postoperative Benefits

P. E. IRELAND, M.D., Toronto
Surgical Emergencies in Laryngology

ROBERT H. IVY, M.D., Philadelphia
Pathological Conditions of the Mouth of Interest to the Laryngologist

CHEVALIER JACKSON, M.D., and
CHEVALIER L. JACKSON, M.D., Philadelphia
I. Benign Growths of the Larynx
II. Cancer of the Larynx

GEORGE L. KING, JR., M.D., Alliance, Ohio
The Differential Diagnosis of Cough

SAMUEL J. KOPETZKY, M.D., New York
The Diagnosis, Differential Diagnosis and Surgical Treatment of Suppurative Petrositis

ROMEO A. LUONGO, M.D., Philadelphia
The Fronto-Ethmo-Sphenoidal Operation

PAUL B. MacCREADY, M.D., New Haven, Conn.
Operative Treatment of Sinusitis

E. M. MacEWEN, M.D., Iowa City (by invitation)
Applied Anatomy of the Neck

A. S. MACMILLAN, M.D., Boston (by invitation)
X-Ray Examination of the Sinuses and Mastoids

C. H. McCASKEY, M.D., Indianapolis
The Diagnosis and Treatment of Affections of the Neck Which Are Related to Otolaryngology

WILLIAM J. MELLINGER M.D., Santa Barbara, Calif.
The Venous Circulation of the Head and Neck and Its Clinical Importance

PAUL M. MOORE, JR., M.D., Cleveland
Some Problems of Office Practice and Their Treatment

H. P. MOSHER, M.D., Boston
Esophagus

HORACE NEWHART, M.D., Minneapolis
Audiometers and Hearing Aids, Their Scope, Limitations and Practical Application

SAMUEL J. PEARLMAN, M.D., Chicago
Deep Infections of the Neck

LYMAN RICHARDS, M.D., Boston
Diagnosis and Practical Treatment of Sinus Disease in Children

JAMES MILTON ROBB, M.D., Detroit
Tonsillectomy and Adenoidectomy and Their Complications

HARRY P. SCHENCK, M.D., Philadelphia
The Management of Pharyngeal Lymphoid Hyperplasia

JOHN J. SHEA, M.D., Memphis
The Treatment of Acute Infections of the Nose and Throat

BENJAMIN H. SHUSTER, M.D., Philadelphia
Vertigo

FERRIS SMITH, M.D., Grand Rapids, Mich.
Some Principles of Plastic Surgery of the Face, Nose and Eyelids

LAVERNE B. SPAKE, M.D., Kansas City, Kan.
The Present Conception of Hemorrhage Control

CLAIRE L. STRAITH, M.D., Detroit (by invitation)
Plastic Surgery of the Face

GABRIEL TUCKER, M.D., Philadelphia
I. Present Status of Bronchoscopy in the Diagnosis and Treatment of Lower Respiratory Disease
II. Gastroscopy for Diagnosis of Disease and the Removal of Foreign Bodies from the Stomach

HARRIS H. VAIL, M.D., Cincinnati
Headaches and Neuralgia of the Face Encountered by the Otolaryngologist

O. E. VAN ALYEA, M.D., Chicago
Irrigation of the Frontal and Maxillary Sinuses Through Their Natural Openings

R. PERCY WRIGHT, Montreal
Atrophic Rhinitis and Ozena

Motion Pictures
and
Instruments

The American Academy
of Ophthalmology and Otolaryngology

Forty-Third Annual Meeting
Washington • *1938*

Motion Pictures and Instruments

SECRETARIES IN CHARGE

W. L. BENEDICT, M.D., *for Ophthalmology*

J. L. MYERS, M.D., *for Otolaryngology*

* * *

Mechanics.....BELL & HOWELL COMPANY

* * *

OPHTHALMOLOGY

Presiding: WEBB W. WEEKS, M.D., *Second Vice President*, New York

A. *Extracapsular Cataract Surgery* (color)

B. *Intracapsular Cataract Surgery* (color)

D. B. KIRBY, M.D., New York

External Orbitotomy. Motion Picture Demonstration (in color)

E. B. SPAETH, M.D., Philadelphia

Cataract Extraction Through Vertical Conjunctival Slit (in color)

WALTER MOEHLE, M.D., Brooklyn

A. *Plastic on Eyelid for Epithelioma*

B. *Arachnodactally*

WENDELL L. HUGHES, M.D., Hempstead, L. I., N. Y.

A Galvano-Diathermic Micro-Puncture Treatment of Separated Retina with Macula Hole Present or Anticipated, with a New Device to Warn Patients of Eye Movements

CLIFFORD B. WALKER, M.D., Los Angeles

A. *Tattooing of the Cornea with Gold Chloride*

B. *Enucleation with Gold Sphere Implantation*

C. *Keratoplasty. Trephine Technique*

RAMON CASTROVIEJO, M.D., New York

Conditions Governing Iris Prolapse After Cataract Operation

ANDERSON C. HILDING, M.D., Duluth, Minn.

Orthoptic and Surgical Treatment of Strabismus

R. K. DAILY, M.D., Houston, Texas

A. *Repair of Cicatricial Ectropion*

B. *Enucleation of Eyeball*

C. *Intracapsular Cataract Extraction*

D. *Dacryocystorhinostomy*

FRANK BARBER, M.D., Rochester, N. Y.

Surgical Relief of Ptosis Associated with Jaw Winking Phenomenon

DESMOND CURRAN, M.D., Kansas City, Mo.

The Flourescent Lamp in Surgery of the Lens

H. ROMMEL HILDRETH, M.D., St. Louis, Mo.

OTOLARYNGOLOGY

Presiding: JOHN H. FOSTER, M.D., *Third Vice President*, Houston, Texas

A group of Academy members, under the coordination of Secretary Myers, have been creating a film which it is hoped will be available for teaching purposes. The title . . .

"METHODS OF EXAMINING THE LARYNX"

AUTHORS

Indirect Laryngoscopy .....JOHN L. MYERS, M.D.

Indirect Laryngoscopy
with Stroboscope .....LYLE S. POWELL, M.D.

Direct Laryngoscopy with Lynch's
Suspension Method.....FRANCIS E. LEJEUNE, M.D.

Direct Laryngoscopy with Haslinger's
Proximal Lighted Scope....DEAN M. LIERLE, M.D.

Direct Laryngoscopy with Jackson's
Distal Lighted Scope..CHEVALIER L. JACKSON, M.D.

Direct Laryngoscopy with Bruner's Scope
and with Patient in Chair...SAMUEL IGLAUER, M.D.

Anatomy and Physiology
of Larynx .....JOEL J. PRESSMAN, M.D.

Technic of Intranasal Sinus Operations

L. M. HURD, M.D., New York

Amputation of the Nose and External Operations on Frontal and Maxillary Sinuses

M. S. ERSNER, M.D., Philadelphia

Lacrimal Sac Operations

T. S. BLAKESLEY, M.D., Kansas City, Mo.

Acute Frontal Sinus, Osteomyelitis, Brain Abscess, Diagnosis, Treatment, Results

S. R. SKILLERN, M.D. (Coates & Whelan), Philadelphia

Diverticulum of the Hypopharynx (A film showing operation by the one stage esophagoscopically-guided method. Kodachrome; one reel.)

C. L. Jackson, M.D., and
W. Wayne Babcock, M.D., Philadelphia

Stenosis of the Larynx with Special Reference to the Method of Treatment by "Core Moulds"

C. L. Jackson, M.D., Philadelphia

Laryngofissure for Early Carcinoma of the Larynx

Louis H. Clerf, M.D., Philadelphia

Laryngofissure Intrinsic Cancer (color)

Arthur Palmer, M.D., New York

Laryngectomy for Extensive, Intrinsic Cancer of the Larynx

Gabriel Tucker, M.D., Philadelphia

Living Laryngectomized Patients. Instruments of Aid in O. R. L. Diagnosis

J. C. Beck, M.D., Chicago

Radical Mastoidectomy with Skin Graft (in color)

Arthur G. Fort, M.D., and
Lester A. Brown, M.D., Atlanta, Georgia

A NEW PURE TONE AUDIOMETER*

HORACE NEWART, M.D.
MINNEAPOLIS, MINN.

FIG. 1 — (Newhart) — New maico D-6 school audiometer.

SINCE the advent of the phonograph audiometer for simultaneously testing the hearing of school children there has been a need for a simple, well constructed pure tone audiometer at a reasonable price for individually testing for screening purposes the members of large groups of school children, students and industrial workers.

Without claiming any credit for its technical development I present an instrument of this type. Early in 1938 I suggested to Mr. A. E. Allison, the electro-acoustic engineer of a company

* Presented at the Forty-Fourth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Atlantic City, N. J., April 29, 1938; at the Seventy-First Annual Meeting of the American Otological Society, Inc., Atlantic City, N. J., May 6, 1938, and before the Section on Laryngology, Otology and Rhinology at the Eighty-Ninth Annual Session of the American Medical Association, San Francisco, June 17, 1938.

manufacturing a widely distributed audiometer for diagnostic purposes, the growing demand for a simplified instrument for the rapid but accurate detection of hearing deficiencies, without regard to diagnosis. He at once recognized the large potential demand and within three weeks produced this audiometer known as the Maico D-6 school audiometer (Fig. 1).

It is a pure tone audiometer with a high grade air conduction receiver, silent tone interrupter and push button signal light. Its operating range covers seven frequencies at octave intervals from 128 to 8192 cycles, with intensities variable in steps of 5 decibels. It is operated by the 110 volt 60 cycle electric current.†

Range of Intensity of the New Pure Tone Audiometer

Test Tone (Cycles per Second)	Range of Intensity (Decibels)
128	70
256	80
512	100
1024	100
2048	100
4096	95
8192	80

The instrument is accurately calibrated and practically meets, as nearly as other audiometers submitted, the tentative minimum requirements of the American Medical Association Council on Physical Therapy for "acceptable" audiometers as far as they have been formulated and are applicable to instruments of this kind.

Its outstanding features are the uniform zero reference level and the easily read, illuminated frequency and intensity control dials which make possible rapid and accurate operation. For screening purposes an average of from 30 to 50 individuals can be checked per hour under favorable conditions, or from 150 to 250 in a school day. Pupils too young to be tested by the phonograph audiometer, which requires the ability to write dictated numbers, are easily tested individually. Retests of those discovered to have significant deficiencies can be quickly recorded on a form showing the degree of loss (Fig. 2).

† Battery operated models at additional cost for use in rural areas without electric current will be available.

Name..... *Date*.....

D. B. Loss	128	256	512	1024	2048	4096	8192	D. B. Speech Loss
R. Ear								
L. Ear								

FIG. 2 — (Newhart) — Form for recording the hearing loss.

These features and the low cost ($130.00) make audiometers of this type especially suitable for use in public schools and in the student health services of colleges and universities. They will prove of great usefulness in public health work and in industrial medicine for disclosing hearing defects which frequently are overlooked when the more familiar, cruder methods are employed.

Since audiometers of this type do not provide the means for testing by bone conduction and have no masking device they are not adapted for diagnostic purposes or for prescribing hearing devices. Thus the nurse or technician is not so likely to yield to the frequent request to make a diagnosis and so encroach upon the field of the medical practitioner.

One audiometer of this type should adequately serve at a minimum cost the needs for screening purposes of a school system, college or industrial group containing up to 3,000 or 4,000 individuals. While the technic is simple and can easily be applied by a nurse, technician or teacher, the work should be carried out and the findings evaluated under the supervision of an otolaryngologist or qualified physician.

TEMPORAL BONE CORER AND EXTRACTOR

JOSEPH C. BECK, M.D.
CHICAGO

THESE instruments were devised because of the fact that many people objected to complete autopsies about the head. With these instruments the author felt that the temporal bone could be removed postmortem by means of a simple mastoid incision and thus the head of the corpse would not be distorted in any way.

The temporal bone corer is really a large trepine operated with a hand brace. In the center of the trephine is a small bit also attached to the brace. This bit extends out from the trephine for one-half inch and acts as a guide for the path of the trephine. The bit is removed by turning a hand screw on the side of the trephine as shown in the illustrations.

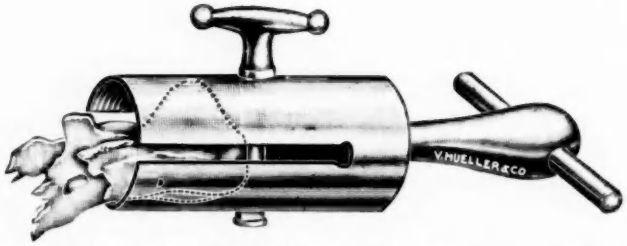


FIG. 1 — (Beck) — Dr. F. Lowell Dunn's Trephine as used by Dr. Joseph C. Beck for the removal of the temporal bone.

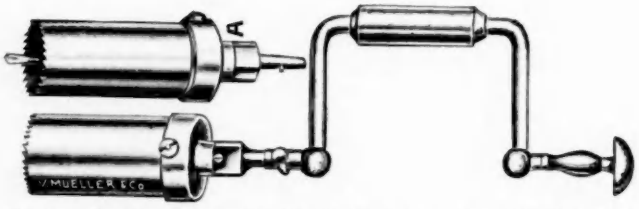


FIG. 2 — (Beck) — Dr. Joseph Beck's Instrument for Removing Temporal Bone after being cut with Dr. Lowell Dunn's Core Drill.

The trephine is made in two sizes — one for adults and one for children. The adult size is 3¾ inches long and 2 inches in diameter. The children's size is 3¾ inches long and 1⅝ inches in diameter.

The temporal bone extractor as shown in the illustration is a bivalve cylinder 2 inches in diameter and 4 inches long and has a hand screw on the side which when turned causes the two blades of the instrument to approximate each other thus holding the temporal bone tight between them. By several lateral movements of the extractor the temporal bone is removed together with some soft tissue attached.

The corer is used in this way: After a post auricular incision over the mastoid is made and the periosteum overlying the bone is incised and pushed aside, the bit is placed about 4 mm. inferior and posterior to the bony canal and the direction is forward, superior and anterior. In some cases a horizontal incision must be made across the middle of the longitudinal postauricular incision in order to have more room. After several turns with the brace the bit is removed and the trephine is set in motion in the same direction. At this time the auricle is pulled forward and the instrument severs the cartilaginous canal from the bony canal. After the trephine was traversed its entire length it is then removed and the extractor placed in position and the temporal bone is removed.

The defect in the skull is filled with plaster of paris and the overlying skin sutured in the usual manner. This procedure can be done on both embalmed and unembalmed specimens alike without interference of the embalming process. The temporal bone specimen removed in this way contains all the essential anatomical details that are necessary for study.

A NEW SURGICAL NEEDLE AND HOLDER

FRANCES RICHMAN, M.D.
BROOKLYN

THIS needle and holder were devised to take the place of the ordinary needle and holder with a view to eliminating the disadvantages of the latter, especially when used in ocular surgery.

Unlike the ordinary surgical needle, this one is firmly anchored to the handle to form a single unit or instrument; when, however, it is necessary to change the type of needle, it can be readily detached and replaced by any other designated to meet the needs of the specific case. Handles, too, are interchangeable in like manner.

This needle has two eyes, one near the point for carrying the suture, and another further back for guiding the suture, with a groove along the undersurface connecting the two, as is shown in the photograph (Fig. 1).

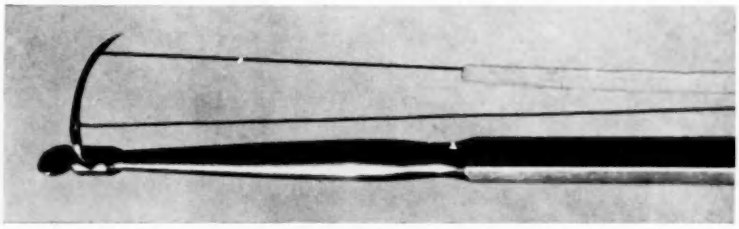


FIG. 1 — (Richman)

Under surface of suture-placing needle showing relationship of the two eyes and connecting groove. The black silk suture was placed on the stretch for better definition.

The several steps in the technique of suturing by this new method are shown in the accompanying sketch (Fig. 2). Only the tip of this suture-placing needle needs to be inserted through the lips of the incision; as soon as the loop of suture threaded in the tip eye appears in view it is grasped and held while the needle is withdrawn. If a continuous suture is desired, it is only necessary to pass a continuous locking thread through each loop as it is placed, then to draw the loop sufficiently taut to procure a good

approximation of the lips of the wound. Continuous sutures are easily removed by pulling out the locking thread that holds them. If knotless interrupted sutures are preferred, simply draw the two cut ends through the loop twice; this will result in a secure closure without any tension on the tissues. This method is also useful for placing mattress sutures as it obviates the need for double-armed sutures. Other combinations of stitches to suit the requirements of each case will become apparent to the surgeon as he familiarizes himself with the possibilities of this instrument. Aside from this, it eliminates the disadvantages of ordinary suturing such as the repeated positioning of the needle in the jaws of the holder, the repeated releasing of the needle from the jaws of the holder, the repeated grasping of the tip of the needle in order to pull it through th tissues, the traumatization of tissue by drawing the full length of suture material through at each bite, and so forth.

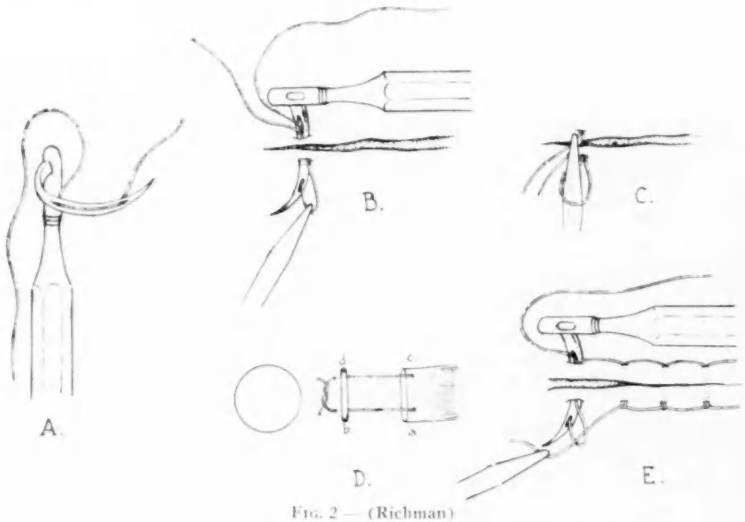


FIG. 2 — (Richman)

A. Shows the suture-placing needle threaded and ready to use.

B. Shows the needle passed through the two lips of the incision and the loop grasped preparatory to withdrawing the needle.

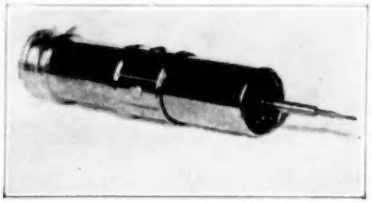
C. Shows a suture in place with the ends being drawn through the loop for locking.

D. Shows a simple method of placing a mattress suture in eye muscle surgery by pulling out short end of both loops and tying.

E. Shows a method of placing a continuous suture. Three sutures have already been placed and locked, the fourth suture shows the locking thread being drawn through the loop preparatory to withdrawing the needle.

BATTERY-DRIVEN CORNEAL BURR

EMANUEL KRIMSKY, M.D.*
BROOKLYN



NOT LONG before his death, Dr. Isadore Goldstein requested me to construct for him a few things, and this instrument is one of his suggestions.

The construction of this device was a rather simple matter. An ingenious battery-driven revolving fan, made on the continent, was deprived of its blades and, in place of them, a cylindrical collar was attached to which the operator could readily insert any of the standard dental burrs. The handle itself is a rather compact unit, consisting of a generator operated by two batteries, the entire outfit occupying surprisingly little space. The speed of this motor was found to be satisfactory for general requirements — about 500 revolutions per minute — and could be readily reduced by the simple application of the index finger to the projecting rod. The whole unit can easily be held in the hand.

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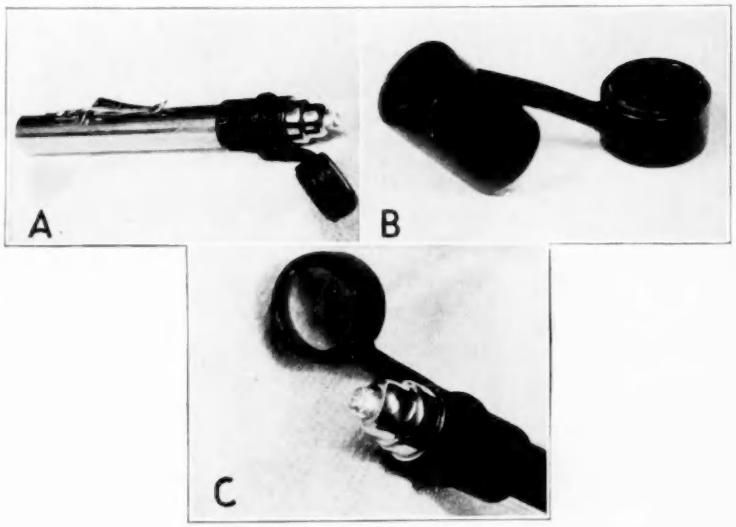
* As suggested by the late Dr. Isadore Goldstein.

FOCUSSING MAGNIFIER ATTACHMENT

EMANUEL KRIMSKY, M.D.
BROOKLYN



BECAUSE of the added advantages of the new penlight lamp with its new style solid glass bulb which renders a concentrated beam of light, the author has found it useful to construct a magnifying lens attachment which would enlarge its scope beyond that for which it was intended.

For this I have had devised a simple unit consisting of a collar through which the flashlight can be easily slipped, a connecting bracket, and a lens mount which has incorporated in it a 6× double aplanatic lens. This attachment sets firm around the battery handle. The lens is so tilted that its focal point meets the beam of light. The entire unit is firm, light, and, moreover, breakage of flashlight, as so frequently happens, permits the attachment to be readily transferred to a new lamp with but very little additional cost. The bracket is firm and inflexible.

DESCRIPTIVE ATLAS OF ORTHOPTIC SLIDES

EMANUEL KRIMSKY, M.D.
BROOKLYN

IN PREPARING a set of pictures for use with my Senior Phorometric and Junior Companion Stereoscopes, I felt that a description of their purposes and uses might convey some meaning to those of us who are as yet unfamiliar with the stereoscope and its mysterious pictures. And so, I have attempted to produce a descriptive atlas which shall explain not only the meanings of all these slides, but also as to how and why they should be used in the stereoscope.

The atlas proper consumes but a minor portion of the lengthy text, comprising a number of as yet unpublished articles on the subject of orthoptics. The table of contents immediately following the atlas includes such chapters as:

1. The Meaning of Fusion
2. The Nature of Stereoscopic Slides
3. The Retinal Angles Which Targets Subtend
4. The Meaning of Macular and Paramacular Suppression
5. Primary Positions of the Eyes with Reference to Stereoscope Targets
6. How the Primary Position Is Affected by the Lenticular Separation of Viewing Lenses
7. How to Increase the Adduction Range with the Stereoscope
8. The Measurements of Vertical Phoria and Vertical Ductions
9. Stereoscope Study of a Case of Squint
10. Some Physiological Considerations in Orthoptic Training
11. Some of the Advantages of a Stereoscope
12. How to Record Stereoscope Findings
13. Stereoscope Case Records Showing Its Value in Diagnosis
14. Practical Considerations in Orthoptic Training, etc., etc.

The pictures are classified not according to Grades 1, 2, and 3, but as to whether they are dissimilar, non-stereoscopic fusion slides, and stereoscopic slides. They have been designed with espe-

cial regard for the needs of the young, restless, and illiterate child. Simplicity has therefore been the keynote in this project. The plates have been numbered to correspond to the actual plates used with the stereoscope for purposes of ready reference. The slides have been, as far as possible, critically analyzed in the detailed text which follows this slide presentation.

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Krimsky, E.: *The Stereoscope in Theory and Practice, also A New Precision Type Stereoscope.* Brit. Jour. Ophthalmology, April, 1937.

Krimsky, E.: *Some Newer Developments in Precision Type Stereoscopes.* Arch. of Ophthalmology, March, 1938.

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A FEATHERWEIGHT SLIT-LAMP

EMANUEL KRIMSKY, M.D.
BROOKLYN

IN THE August, 1932, issue of the Archives of Ophthalmology, I reported an illuminated loupe. I stated at that time: "One of the difficulties confronting the ophthalmologist in the use of the monocular loupe is in obtaining a sufficient amount of condensed light at a suitable focal distance. Besides the fact that one hand is occupied in holding the condensing lens, the source of the illumination may not always be close or conveniently at hand. In order to overcome these shortcomings it occurred to me that if the light source and condenser could be combined as a unit and this in turn attached or fastened to the loupe, a satisfactory and reliable source of illumination might be effected to assist the operator in his observations." In this article I described how this crude model could also be used as a hand slit-lamp.

In designing a slit-lamp for hand use, my problem was not merely to provide me with a so-called featherweight instrument that could be carried in the hand, but one which would not interfere with the free use of the ophthalmoscope. Hence, besides being a light instrument, it had to be independent in the sense that the use of a common battery handle might prove time-consuming, and expose these articles to breakage through such frequent interchange. I have therefore found it practical to respect the intactness of the ophthalmoscope with its battery handle, and to honor the slit-lamp with a separate medium-sized battery handle (carried in the coat pocket), using No. 935 batteries and connected to the slit-lamp by means of fine silk-woven wire. Such an arrangement permits the slit-lamp to be slipped back into the coat pocket, adds to its lightness by not being joined directly to the handle, obviates frequent disturbance of the ophthalmoscope, is time-saving, and permits it to be held between two fingers instead of by two hands. Furthermore, as will be shown later, by the attachment of a spring clamp to the wearer's eyeglass both hands may be entirely free to permit use of instruments in such conditions as the removal of foreign bodies, or where the operator's hands demand other uses.

FIG. 1 — Slit-Lamp Proper

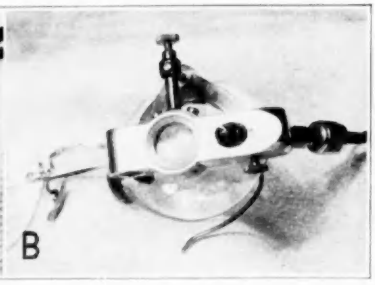

FIG. 2 — With attachment

This new slit-lamp like its forerunner has been designed to include the following features: (1) Centralization and proper angulation of light at an angle of 45°, (2) firmness, (3) lightness, (4) compactness and simplicity of operation, and (5) proper quality and quantity of illumination. In addition, the newer features are: (1) Automatic device to change beam progressively from a vertical slit, then a transverse slit, and finally a homogeneous circle of light, (2) attachment to permit it to be worn over eyeglass, (3) a slit-lamp unit, rather than a modified loupe.

Let us review the features in more detail:

First, The Slit-Lamp Proper — Consists of a lens housing made of duraluminum, at one end of which is a lens mount containing an improved Zeiss 10× double aplanatic lens, and at the other, a channel which makes a 45° angle with the focal axis which holds the lamp unit.

Second, The Lamp Unit — Consists of a light carrier a straight filament bulb, and a hemispherical condenser. This unit has been made to automatically and progressively provide a vertical slit, a horizontal slit, as well as a homogeneous circle of light by the simple turning of the base of this unit which has had incorporated in it a helical mount which operates in such a way as to slide the bulb backwards or forwards without displacing the condenser. By such manipulation, the approximation of bulb and condenser produces a circle of light, and separation leads to a slit light through bringing the filament in the focal plane of the condenser.

Third, Weight of Instrument — The slit-lamp proper including the light carrier weighs but 29 grams (less than one ounce). The addition of the spring clamp brings its weight up to 37 grams.

However light this unit is, it can be made still lighter by the use of plastic materials.

Fourth, The Spring Clamp — Consists of a pair of clamps so constructed that the eyeglass can be slipped between them and held in place. This clamp has at its upper part a horizontal rod which extends back to meet a vertically projecting rod that joins the slit-lamp proper. When not in use, this clamp can be easily freed by means of a fastening screw.

Fifth, Centralization and Proper Angulation of Light — A fixed angulation of the light carrier at 45° to the axis of the lens was found most desirable for practical requirements.

Sixth, Lens Used — While a 10× Zeiss lens was used in this instrument, it would be preferable in cases where attachable clamp is worn frequently, to have a weaker glass (8×) which increases the focal length or distance from the eyes sufficiently to allow freer use of the hands.

This entire unit can be easily disassembled. Moreover, by removal of lamp unit from lens housing proper, the operator can by manipulating the condenser, adjust the slit to satisfy his particular requirements.

REFERENCE

Krimsky, E.: *A Modified Monocular Loupe*. Arch of Ophthalmology, August, 1932, vol. 8, pp. 272-274.

INSTRUMENTS FOR USE IN LARYNGOFISSURE

MERVIN C. MYERSON, M.D.
NEW YORK

THESE instruments are being presented because they have proven to be of value in simplifying the operation of Laryngofissure for cancer of the larynx. The special instruments used are:

1 — A special rotary saw blade.
2 — A pair of three-pronged sharp rectractors.
3 — A special elevator.
4 — A pair of double ended, blunt retractors.
5 — A heavy, retangular punch.
6 — Straight and right angled hemostats.
7 — Curved angular cutting scissors.
8 — Angular knife for cutting through cartilage.

The saw blade is approximately 6 mm. in diameter. It is designed for use with a special hand motor and is employed for cutting through the center of the thyroid cartilage with facility and precision. When used, the blade penetrates the cartilage but does not cut through the internal perichondrium and soft tissues.

The elevator is used for two purposes: To ascertain whether the cartilage is completely severed; to elevate the internal or external perichondrium.

The three-pronged sharp retractors are insinuated between the two halves of the thyroid cartilage, between the cartilage and perichondrium. When so placed they will not tear the cartilage. These retractors are used to separate the two halves of the cartilage, so that the soft tissues may be incised.

The double retractors have, at one end, a rounded blade similar to an eyelid retractor, and at the other end a flat blade at a right angle to the shaft which is quite similar to the Freer submucous retractor. The rounded end is used for retraction of the alae. To accomplish this it is placed upon the lateral aspect of the thyroid notch, after the anterior soft tissue structures have been

FIG. 1 — (Myerson) — A special rotary saw blade.

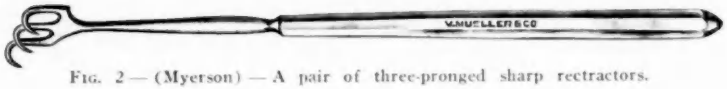

FIG. 2 — (Myerson) — A pair of three-pronged sharp rectractors.

FIG. 3 — (Myerson) — A special elevator.

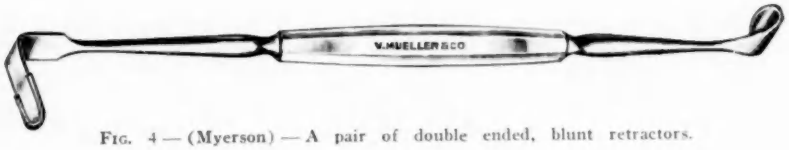

FIG. 4 — (Myerson) — A pair of double ended, blunt retractors.

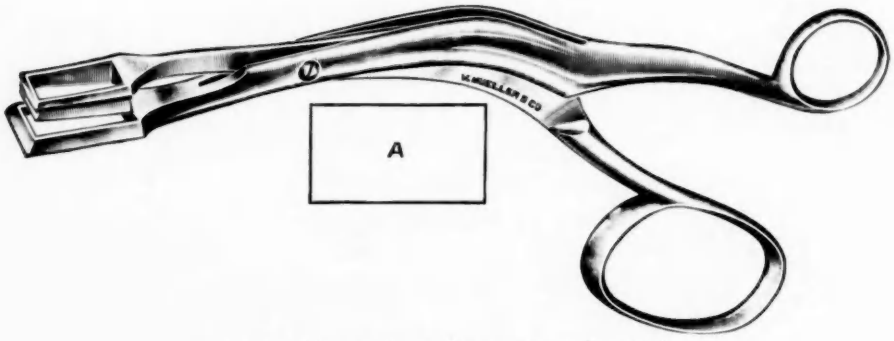

FIG. 5 — (Myerson) — A heavy, rectangular punch.

FIG. 6 — (Myerson) — Straight and right angled hemostats.

FIG. 7 — (Myerson) — Curved angular cutting scissors.

FIG. 8 — (Myerson) — Angular knife for cutting through cartilage.

cut through. This placement of the retractors keeps them out of the field of operation. The flat end is used for retracting the external perichondrium and soft tissues away from the cartilage, so as to facilitate whatever instrumentation may be required.

The punch is a specially heavy instrument designed to crush the tissues in the line of excision of the cartilage and other structures which bear the carcinoma. Whenever possible the punch removes these structures with a single stroke. If the cartilage should be ossified and difficulty is encountered, the tissues are first crushed by the punch, after which straight and specially curved scissors are used to cut through the crushed area. This insures hemostasis. The punch removes a piece of tissue approximately 14 by 24 mm.

The straight and right angled hemostats are used to crush the soft tissues preliminary to excision when the underlying cartilage is to be separately removed.

The scissors curved at an angle are used for cutting from above downward in order to sever the soft tissue, cancer bearing structure when it has already been detached above and below.

The angular knife is used for cutting through the cartilage from above downward when the cartilage is to be removed as a preliminary to excision of the cancer bearing area.

Necrology

The American Academy
of Ophthalmology and Otolaryngology

Forty-Third Annual Meeting
Washington 1938

IN MEMORIAM

James Henry Andrew, Brooklyn, New York
Claude Spencer Beebe, Milwaukee, Wisconsin
J. Paul Ernest Bousquet, Montreal, Canada
Edward Josiah Brown, Minneapolis, Minnesota
Howard Sherwood Busler, Lansdowne, Pennsylvania
Theodore Albinus Coffelt, Powersite, Missouri
William Edward Colgin, Waco, Texas
Claude E. Cooper, Denver, Colorado
Lewis Webb Crigler, New York, New York
Charles Ludvey Davis, San Diego, California
John Dodds Flagg, Buffalo, New York
Harry Burton Fuller, Chicago, Illinois
Charles Neumann Gelber, New York, New York
Isadore Goldstein, New York, New York
George F. Gourley, Steubensville, Ohio
F. J. Hackney, Chattanooga, Tennessee
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Alfred Lubeert Van Dellen, Chicago, Illinois
John Martin Wheeler, New York, New York
Charles Peter White, Wilmington, Delaware
John Robert Wilson, Bennington, Vermont
Harry Vanderbilt Wurdemann, Seattle, Washington

James Henry Andrew, M.D.

Dr. Andrew was born in Cambridge, N. Y., on July 29, 1874, and died in Brooklyn, N. Y., on November 26, 1937.

After preliminary education at Brooklyn Polytechnic Institute, he received his medical degree from the Bellevue Hospital Medical College, New York, in 1896, and began the practice of medicine in Brooklyn in 1897.

Dr. Andrew was a specialist in diseases of the eye and at the time of his dealth was Ophthalmologist at St. John's, Bushwick, Samaritan and Shore Road Hospitals, Surgeon at the Brooklyn Eye and Ear Hospital, and Consulting Ophthalmologist at the Beth Moses and Huntington Hospitals.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and of local, state and Special Medical Societies.

He is survived by his widow and three sisters.

Claude Spencer Beebe, M.D.

Dr. Beebe was born in Evansville, Wisconsin, on December 27, 1877, and died in Milwaukee, Wisconsin, on May 24, 1938.

After being graduated from the University of Wisconsin, he obtained his medical degree at the Wisconsin College of Physicians and Surgeons in 1904, and took post-graduate work in New York at Dr. Herman Knapp's Eye Clinic and at Moorfield Hospital in London, England. He began the practice of medicine in Milwaukee in 1905, in partnership with his father, who was an eye, ear, nose and throat specialist. Following the death of his father in 1915, Dr. Beebe devoted his time almost exclusively to diseases and injuries of the eye, and developed a practice devoted very largely to industrial eye injuries, due to Milwaukee being such a diversified industrial center. While a member of the Health Board in 1924-25 and also on the School Board of Shorewood he became especially interested in the prevention of disease in the children of school age and took very forward steps in developing the modern method of examining school children to check the spread of contagion. At one time he was Instructor of Bacteriology at the Wisconsin College of Physicians and Surgeons.

At the time of his death Dr. Beebe was on the staff of the Milwaukee Hospital, the Milwaukee Children's Hospital and the Johnson Emergency Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the Royal Ophthalmic Society of London, England, and of local and state medical societies.

He was an enthusiastic photographer and a great traveler.

His wife, two sons and two daughters survive him.

J. Paul Ernest Bousquet, M.D.

Dr. Bousquet was born in St. Denis-sur-Richelieu, P. Q., on January 4, 1881, and died in Montreal, P. Q., on October 27, 1937.

He was educated at St. Hyacinthe Seminary, and graduated with highest honors from Laval University Medical School, Montreal, in 1906. He studied ophthalmology in Paris for several years, and became a member of the Societe Francaise d'Ophthalmologie de Paris in 1909. Later he returned to Montreal and St. Hyacinthe to take up the practice of medicine, specializing in diseases of the eye, ear, nose and throat.

He was Titular Professor of Ophthalmology at the University of Montreal and ophthalmologist-in-chief at the Hotel Dieu Hospital.

Dr. Bousquet was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, an officer of the French Academy since 1925, President of the Societe d'Ophthalmology de Montreal, Corresponding Member of the Societe Francaise d'Oto-rhino-laryngologie de Paris and of the Societe Belge d'Ophthalmologie, and member of local and provincial medical societies.

He represented the Province of Quebec and the University of Montreal in 1925 at the Congress in Brussels of the Societe Francaise d'Ophthalmologie de Paris, and in 1929 again filled this role at the Ophthalmo-oto-neurologie Societe at Bordeaux, France.

He is survived by his wife, three daughters, one son, three brothers and two sisters.

Edward Josiah Brown, M.D.

Dr. Brown was born in Burke, Vermont, on January 14, 1851, and died at Minneapolis, Minnesota, on March 15, 1938.

After obtaining his preliminary education at Kimball Union Academy, Hanover, N. H., he obtained his medical degree from

Dartmouth Medical College, and did post-graduate work at Columbia Medical School, and in the clinics of Berlin and London. He began to practice medicine in Littleton, later moving to Haverhill, New Hampshire, and then to Minneapolis, where he settled.

Dr. Brown at one time was Professor of Preventive Medicine, and later Professor of Diseases of the Eye and Ear, in the College of Physicians and Surgeons, Minneapolis.

He was a life member of the American Academy of Ophthalmology and Otolaryngology, member and past President of the Minnesota Academy of Ophthalmology and Otolaryngology, member and past President of the Hennepin County Medical Society.

Dr. Brown was a frequent contributor to medical and scientific journals, and greatly interested in preventive medicine, being active on city and state Boards of Health.

He is survived by his wife, two daughters, four sons, a brother and two sisters.

Howard Sherwood Busler, M.D.

Dr. Busler was born in Philadephia, Pa., on December 11, 1888, and died in Lansdowne, Pa., November 16, 1937.

He received his medical degree from Hahnemann Medical College in 1911, and began the practice of medicine in Lansdowne in 1912 following an interneship at the Buffalo General Hospital. In 1927-28-29 he did post-graduate ear, nose and throat work, in the clinics of Vienna and Budapest, and later confined his medical work to those specialties.

At the time of his death, Dr. Busler was Instructor in Laryngology and Rhinology at Hahnemann Medical College, chief of staff at Fitzgerald-Mercy Hospital, and director of the ear, nose and throat clinic at Delaware County Hospital, Drexel Hill, Pa.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, and member of local and state medical societies.

Dr. Busler served in the Army Medical Corps during the World War.

His widow, a daughter, his parents and a brother and sister survive him.

Theodore Albinus Coffelt, M.D.

Dr. Coffelt was born in McDonald County, Missouri, on April 10, 1855, and died in Powersite, Missouri, on September 5, 1937.

He received his medical degree from the Missouri Medical College, St. Louis, 1886, and began the practice of medicine in Benton County, Arkansas. Later he took post-graduate work at the New York Post-Graduate School of Medicine, and moved to Springfield in 1893 for the practice of his specialties. He had retired from active practice several years ago. He was formerly on the staff of the Springfield (Mo.) Hospital.

Dr. Coffelt was a member of the American Academy of Ophthalmology and Otolaryngology, and at the time of his death President of the Taney County Medical Society.

He was very much interested in fruit farming, and for a number of years had been a minister in the Methodist Church.

His wife, one brother and five children survive him.

William Edward Colgin, M.D.

Dr. Colgin was born in Waco Texas, on July 6, 1900, and died of accidental electrocution while repairing an electric connection at his home there on September 10, 1937.

He received his academic education at the Columbia Military Institute, Columbia, Tennessee, and the University of Texas, Austin; his medical education at Loyola Medical College, Chicago, from which he was graduated in 1928. After an interneship at the Alexian Brothers' Hospital in Chicago, and a residency at the Illinois Eye and Ear Infirmary, Chicago, he returned to Waco and entered practice with his brothers, taking charge of the eye, ear, nose and throat department of the Colgin Hospital and Clinic.

At the time of his death Dr. Colgin was assistant medical director of the Texas Life Insurance Company, Division Eye, Ear, Nose and Throat Surgeon for the M. K. & T. Railway Company, and local surgeon in the same capacity for the Missouri Pacific Lines.

Dr. Colgin was a member of the American Academy of Ophthalmology and Otolaryngology; of his state and local medical societies, and had been recently elected Vice-President of the Dallas Ophthalmological Society.

He took an active interest in child welfare work and in life insurance. His hobby was electricity.

His wife, two young sons, his mother, a brother and sister, survive him.

Claude E. Cooper, M.D.

Dr. Cooper was born in Denver, Colorado, on November 27, 1878, and died there on June 21, 1938.

He received his early education at Regis College, Denver, and Georgetown University, Washington, D. C., and his medical degree from Gross Medical College, Denver, and in 1900 began the practice of medicine in his home town.

Dr. Cooper was Professor of Otolaryngology at the University of Colorado and on the staffs of the National Jewish Hospital, National Swedish Hospital, Mery Hospital (of which he was one of the founders), St. Joseph's, Children's and Denver General Hospitals.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, and of local and state medical societies, holding official positions in several of them.

Dr. Cooper served during the Spanish-American War, being assigned to the medical corps hospital service in the Philippine Islands.

His wife and two sons survive him.

Lewis Webb Crigler, M.D.

Dr. Crigler was born in Crawford, Mississippi, on June 26, 1876, and died in New York City on April 30th, 1938.

He graduated from Mississippi State College, Starkville, Miss., and obtained his medical degree from Vanderbilt University, Nashville, Tenn., after which he took post-graduate eye work at the Manhattan Eye, Ear & Throat Hospital, New York City. He began the practice of medicine in 1899 at Aberdeen, Miss., later moved to Columbus, Miss., and then to New York City in 1909, at which time he specialized in diseases of the eye.

At the time of his death Dr. Crigler was Surgeon Director of the Manhattan Eye, Ear & Throat Hospital, Attending Ophthal-

mologist to the New Rochelle Hospital, and Consulting Ophthalmologist to the Elizabeth Horton Memorial Hospital, Middletown, N. Y., and the United Hospital at Port Chester ,N. Y.

He was a member of the American Academy of Ophthalmology Otolaryngology and local and state medical and special societies.

His widow and two daughters survive him.

Charles Ludvey Davis, M.D.

Dr. Davis was born on March 11, 1884, and died in Pasadena, California, on July 25, 1937.

He was graduated from the St. Louis University School of Medicine in 1908, and began the practice of medicine in Robinson, Ill., in 1908. Later he specialized in ear, nose and throat work, and at one time was Instructor in Clinical Otolaryngology at Washington University School of Medicine; assistant otolaryngologist to the Barnes Hospital and St. Louis Children's Hospital, St. Louis; surgeon to out-patients, Washington University Dispensary, St. Louis; surgeon to St. Louis City Hospital and consultant to Barnard Skin and Cancer Hospital, St. Louis.

Dr. Davis served during the World War.

He was a member of the American Academy of Ophthalmology and Otolaryngology and the Pacific Coast Oto-Ophthalmological Society.

His wife survives him.

John Dodds Flagg, M.D.

Dr. Flagg was born in Morrisburg, Ontario, on January 16, 1860, and died in Buffalo, N. Y., in July 19, 1937.

After receiving his early education in Morrisburg, he prepared for college at Coburg and St. Catharines, Ontario, was graduated from McGill University Medical School, Montreal, with the degree of M.D., in 1887, and received the degree of L.R.C.P. and S. at Edinburgh University in the same year.

Dr. Flagg took up the practice of medicine in Buffalo, later confining himself to his specialty of eye work. For years he was Professor of Anatomy and Ophthalmology at the old Niagara University, and served as Chief of Staff of the Charity Hospital in the eye, ear, and throat department.

He was a life member of the American Academy of Ophthalmology and Otolaryngology. During his long life he devoted himself to the building up of the Charity Eye, Ear and Throat Hospital, as well as giving his time and energy to civic interests.

His wife died several years ago, and he is survived by a brother and a nephew, both of whom are physicians.

Harry Burton Fuller, M.D.

Dr. Fuller was born in Port Huron, Michigan, on July 22, 1886, and died in Chicago, Ill., on February 4, 1938.

He was a graduate of the University of Chicago, obtained his medical degree from Rush Medical College in 1913, and began the practice of medicine in Chicago in 1915.

At the time of his death Dr. Fuller was on the staff of the Chicago Eye, Ear, Nose and Throat Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the American College of Surgeons, and local and state medical societies.

He is survived by his widow, Dr. Muriel Kling Fuller.

Charles Neumann Gelber, M.D.

Dr. Charles Gelber of 30 East 40th Street, New York City, died on March 3 1938, at the age of 46.

He was a graduate of the Long Island College Hospital in 1913, and was at one time medical director of the Gelber Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology.

Isadore Goldstein, M.D.

Dr. Goldstein was born on December 9, 1879, in New York City and died there on December 23, 1937.

He was graduated from Cornell University Medical School in 1904, completed his interneship at Mount Sinai Hospital in 1906, and practiced in New York City since that date. He took post-graduate work in Columbia University and specialized in diseases of the eye.

Since March, 1924, he had been on the attending staff of Mount Sinai Hospital, serving as adjunct and associate ophthalmic surgeon until 1932 when he became ophthalmic surgeon, and since 1931 an associate in ophthalmic pathology. At the time of his death he was also senior assistant surgeon of the New York Eye and Ear Infirmary, consultant Ophthalmic surgeon at Sydenham and Trinity Hospitals and at Beth Abraham Home for the Aged; instructor in ophthalmic surgery at the Post-Graduate Department of Columbia University Medical School.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and a former Vice-President of the Society of Plastic and Reconstructive Surgery, and various local and state medical societies.

He was particularly known for his work in plastic surgery of the eye and was co-originator of a system of criminal identification through mapping of the retinal pattern of the eye. He organized the Ophthalmology Department of the Hebrew University in Palestine, passing considerable time there in this activity.

He is survived by his wife and one daughter.

George F. Gourley, M.D.

Dr. Gourley was born in Frostburg, Pennsylvania, on January 5, 1879, and died in Steubenville Ohio, on June 28, 1938.

He received his medical degree from Ohio State University at Columbus, Ohio, in 1903, and entered general practice for the United States Coal Company at Bradley, Ohio, later practicing at Smithfield, Ohio. After some years of post-graduate study at Harvard Medical School and the eye, ear and nose clinics in Vienna and other European cities, Dr. Gourley located in Steubenville in 1915.

He was a member of the American Academy of Ophthalmology and Otolaryngology and local and state medical societies.

He is survived by his wife.

Francis J. Hackney, M.D.

Dr. Hackney was born in Friendsville, Blount County, Tennessee on July 5, 1873, and died in Chattanooga, October 29, 1937.

His early education was obtained in the Friendville Academy, Maryville College, National Normal University, the Hospital Col-

lege of Medicine, Louisville, Kentucky, and received his medical degree from the College of Medicine of the University of the South, Sewanee, in 1899.

He began the practice of medicine in Knoxville, and established a private sanitarium there, later taking post-graduate ear, nose and throat work at Jefferson Medical College and Wills Eye Hospital, Philadelphia, the Manhattan Eye, Ear and Throat Hospital, the New York Eye and Ear Infirmary and the New York Post-Graduate School, Johns Hopkins University and the Chicago Eye, Ear, Nose and Throat Hospital. In 1912 he moved to Chattanooga and in 1931 established the McCallie Avenue Infirmary, and was on the staff of the Children's Hospital.

Dr. Hackney was a member of the American Academy of Ophthalmology and Otolaryngology, and of local and state medical societies.

He was a Quaker, a descendant of the Quaker pioneers who settled in Blount County, Tennessee, and was a Trustee and benefactor of Friendsville Academy, the Quaker school of his home town. He devoted much time to social service work through the Civitan Club and to the care of crippled children at the Children's Hospital Clinic.

He is survived by his wife, mother, a son and two sisters.

Elmer Ellsworth Hagler, M.D.

Dr. Hägler was born on May 5, 1863, at Virden, Illinois, and died October 17, 1937, at Springfield, Illinois.

His preliminary education was obtained in the public schools, and previous to studying medicine he taught school for three years. His collegiate and medical education was obtained at the University of Michigan, from which he graduated in 1890. He was at one time Instructor and Demonstrator of ophthalmic and aural surgery and clinical ophthalmology and otology at his alma mater.

In 1893, Dr. Hägler moved to Springfield and began the practice of his specialty, being joined in 1899 by Captain Arthur Lee Hägler, M.D., his brother. At the time of his death, Dr. Hägler was on the staff of St. John's Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, past president of the Sangamon County Medical So-

ciety, Fellow of the Royal Microscopic Society, London, and a member of local and state medical societies.

Dr. Hägler spent a considerable period of his young manhood in military service and was captain of Troop D, First Illinois Cavalry in 1898. During the World War he was a member of the Medical Advisory Board, and District Chairman for the National Council of Defense.

He was keenly interested in civic affairs, and a member of various Masonic bodies.

He is survived by one daughter, one son, and a brother. Another son, who died in 1920, and the present surviving son, also served with distinction in the World War.

Harold Bertram Hedrick, M.D.

Dr. Hedrick was born in Leavenworth, Kansas, on June 8, 1890, and died in Kansas City, Missouri, on February 21, 1938.

After graduating from the University Medical College of Kansas City in 1913, he began the practice of medicine and surgery in 1913. Later he took post-graduate work at the Knapp Memorial Hospital, New York City, the University of Pennsylvania and in Vienna.

At the time of his death he was on the staffs of the Trinity Lutheran, Kansas City General, St. Luke's, Mercy and St. Mary's Hospitals.

He was a member of the American Academy of Ophthalmology and Otolaryngology and local and state medical societies.

Dr. Hedrick served during the World War for twenty-two months, first with the British Expeditionary Force in France and Italy as a lieutenant in the Medical Corps and later joining the American army when the medical corps was organized. He was twice wounded and was awarded the British Military Cross, and was discharged with the rank of Captain.

He was a 32nd Degree Mason.

His wife, his mother, two sisters and a brother survive him.

Sandford Halsey Kinne, M.D.

Dr. Kinne was born December 29, 1884, at Ovid Seneca County, New York, and died on April 13, 1938, at Binghamton, New York.

He received his medical degree from Syracuse University College of Medicine in 1909, later took post-graduate work at the Manhattan Eye, Ear and Throat Hospital, and in Vienna.

Dr. Kinne began the practice of his specialties in Binghamton in 1915, and at the time of his death was otolaryngologist to the Binghamton City and Lurdes Hospitals and Charles S. Wilson Memorial Hospital, Johnson City; consulting otolaryngologist to the Binghamton State Hospital, Ideal Hospital, Endicott, and the Tioga County General Hospital, Waverly.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and local and state medical societies.

He is survived by his wife, two sons and two daughters.

Edwin J. Lent, M.D.

Dr. Lent was born in Picton, Ontario, Canada, on August 17, 1870, and died there on May 31, 1938.

After receiving his preliminary education at Picton he was graduated from the Queen's University Faculty of Medicine, Kingston, Ontario, in 1892, and began the practice of medicine in Lakeville, Indiana. In 1899 he took post-graduate training in New York, and became an outstanding specialist in diseases of the eye, ear, nose and throat. In 1918, in cooperation with fellow specialists, he organized the South Bend Clinic, which he served as President for many years.

At the time of his death Dr. Lent was on the staffs of the Epworth and St. Joseph's Hospitals and Children's Dispensary.

He was a member of the American Academy of Ophthalmology and Otolaryngology and of local and state medical societies.

He served during the World War with the commission as Captain, being stationed at the Walter Reed Hospital in Washington, D.C.

His interests other than medical were civic and Masonic.

He is survived by his widow and two grandsons.

John Gustin Love, M.D.

Dr. Love was born in Hanover, Ill., on May 11, 1876, and died in Sedalia, Missouri, on May 15, 1938.

After being graduated from the Beaumont Medical College in St. Louis and the Marion Sims Medical College now part of the

University of St. Louis, and serving his interneship at the Missouri-Kansas-Texas Hospital, he took a post-graduate course at the Chicago Eye, Ear and Throat College, and began the practice of his specialty in Sedalia. In 1907 he was appointed to the medical staff of the state hospital at Nevada, Missouri, where he remained until 1913.

At one time Dr. Love was on the staff of the John H. Bothwell Memorial Hospital, was a member of the medical staff of the Missouri-Kansas-Texas Hospital and the Missouri Pacific Railroad, and did much work for the Missouri Commission for the Blind.

He was a member of the American Academy of Ophthalmology and Otolaryngology, past President of the Pettis County Medical Society and member of other local medical societies.

He was very much interested in civic matters and an enthusiastic Mason.

His widow and one daughter survive.

James Pleasant Matheson, M.D.

Dr. Matheson of Charlotte, N. C., died in an automobile accident near Hartsville, S. C., on August 5, 1937, at the age of 58.

He was a graduate of the University of Maryland School of Medicine, Baltimore, in 1905, and was otolaryngologist to the Charlotte Eye, Ear and Throat, and Presbyterian hospitals, and the Charlotte Sanatorium.

He was a member of the American Academy of Ophthalmology and Otolaryngology, a member of the American Laryngological, Rhinological and Otological Society, and Fellow of the American College of Surgeons.

He served during the World War.

John Craig McAllister, M.D.

Dr. McAllister was born in Brandy Camp, Pennsylvania, on June 13, 1861, and died in Ridgway, Pennsylvania, April 18, 1938.

He received his medical degree from the College of Physicians and Surgeons (now the University of Maryland), Baltimore, Maryland, in 1889, and took post-graduate courses in eye, ear, nose and throat work at Wills Eye Hospital, Philadelphia, Lon-

don and Vienna. He began to practice medicine in Driftwood, Pennsylvania, moving to Ridgway in 1897, and was head of the Eye, Ear, Nose and Throat Department of the Elk County General Hospital. He was oculist for the Pennsylvalia Railroad and the Baltimore and Ohio Railroad.

He was a life member of the American Academy of Ophthalmology and Otolaryngology, life member of the American Medical Association of Vienna, and member of local and state medical societies.

He is survived by his wife, two daughters and one son.

Alfred Edward Maumenee, M.D.

Dr. Maumenee of Birmingham, Alabama, died on June 5, 1938, at the age of 58.

He was a graduate of the Medical College of Alabama, Mobile, in 1905, and was on the staff of the Hillman Hospital.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, and member of the Medical Association of the State of Alabama.

William Craig Meanor, M.D.

Dr. Meanor was born in Murraysville, Pennsylvania, on June 1, 1872, and died in Edgeworth, Pennsylvania, on September 25, 1937.

After preliminary education at the Greensburg Public Schools and Princeton Preparatory School, he received his medical degree from the University of Pennsylvania Medical School. After postgraduate work at the Manhattan Eye and Ear Hospital in New York, he began the practice of medicine at Beaver, Pennsylvania, in 1908, later specializing in diseases of the eye, and maintaining an office in Pittsburgh.

At the time of his death Dr. Meanor was Chief of the Staff of Ophthalmology at Valley Hospital, Sewickley, Pa.

He was a member of the American Academy of Ophthalmology and Otolaryngology, the Association of Pennsylvania Railroad Surgeons, and local and state medical societies.

During the World War, Dr. Meanor served with the rank of Major in the Medical Corps, at Fortress Monroe, at Mineola, Long Island, and then at Issoudun, in France. He was the first Commander of Philip J. Davidson Post, American Legion, Beaver, Pa.

His wife, two daughters, a son and a sister, survive him.

Edward Lee Meierhof, M.D.

Dr. Meirhof was born in Philadelphia, Pennsylvania, on November 24, 1860, and died in New York City on October 25, 1937.

His early education was obtained in Baltimore and he received his medical degree from the University of Maryland in 1881, following which he began the practice of medicine in Baltimore. Later he moved to New York City and took up the practice of his specialty.

At one time Dr. Meierhof was Professor of the Diseases of the Eye at the Baltimore Medical School, and later on the staffs of the Mt. Sinai Hospital, New York Eye and Ear Infirmary, Sydenham Hospital, and the Institute for Improved Instruction of Deaf Mutes, all in New York City. He retired from active practice in 1926, and spent his later years in travel in the United States, Europe and Africa.

Dr. Meierhof was a life member of the American Academy of Ophthalmology and Otolaryngology, a Fellow of the American College of Surgeons, and a member of local and state medical societies.

He is survived by his widow.

William Frederic Moore, M.D.

Dr. Moore was born August 30, 1881, in Williamsport, Pennslyvania, and died April 25, 1938, in Philadelphia, Pa.

He received his preliminary education in the public schools in Philadelphia and his medical degree from the University of Pennsylvania Department of Medicine in 1905. He went to Vienna for post-graduate courses, later taking up the practice of his specialties in Philadelphia.

He was assistant professor of bronchoscopy and esophagoscopy at the Graduate School of Medicine of the University of

Pennsylvania and at the time of his death was on the staff of the Philadelphia General, Episcopal and Children's Hospitals.

Dr. Moore was a member of the American Academy of Ophthalmology and Otolaryngology, the American Bronchoscopic Society, Fellow of the Philadelphia College of Physicians and member of the Philadelphia Laryngological Society.

He served as Captain in the Navy during the World War, being stationed first at the Philadelphia Navy Yard and then, becoming dissatisfied with inaction, was transferred to the Marines and stationed at Quantico.

He was once President of Beta Chapter of Phi Gamma Delta fraternity, and member of the A.M.P.O. medical fraternity. He is survived by his wife.

Frank Douglas Phinney, M.D.

Dr. Phinney was born on October 3, 1873, in Richibucto, New Brunswick, Canada, and died May 30, 1938, in Cincinnati, Ohio.

He attended Mt. Allison Academy at Sackville, N. B., and the University of New Brunswick at Frederickton, N. B., later obtaining his medical degree at the University of Pennsylvania in 1899. He took post-graduate courses in eye, ear, nose and throat work at the New York Ophthalmic and Aural Institute, and in the clinics of Vienna and London, taking up the practice of these specialties in Cincinnati in 1903.

He was ophthalmologist to the Bethesda Hospital, Director of Ophthalmology at the Cincinnati Convalecent Children's Hospital, and during the summers from 1927 to 1937 inclusive, Dr. Phinney gave his services as ophthalmologist and otolaryngologist to St. Anthony Hospital of the International Grenfell Association, Labrador.

Dr. Phinney was a life member of the American Academy of Ophthalmology and Otolaryngology, a Fellow of the American College of Surgeons, and member of local and state medical societies.

He was very much interested in civic affairs, was a member of the Sons of Colonial Wars, and from 1934 to 1937 served as Governor of the Society of Mayflower Descendants in Ohio.

His wife and one son survive him.

Paul J. Pontius, M.D.

Dr. Pontius was born in Mifflinburg Pa., on June 27, 1867, and died in Philadelphia, Pa., on February 16, 1938.

He received his early education in Bucknell Academy and Bucknell University, and his medical degree from the University of Pennsylvania Medical School in 1891. He also received the degrees of Doctor of Laws and Doctor of Science from Bucknell University, in 1930 and 1932 respectively.

Dr. Pontius began the practice of medicine in Chicago, but later returned to Philadelphia to take up his specialty of the eye. At the time of his death he was ophthalmologist to St. Joseph's Hospital, Philadelphia; Masonic Home, Elizabethtown; consulting surgeon to the Wills Eye Hospital; consulting ophthalmologist to the Philadelphia General and Frankford Hospitals, Shriners' Hospital for Crippled Children, Philadelphia, and Grand View Hospital, Sellersville.

Dr. Pontius was keenly interested and active in Masonic circles and extended this interest to his A.M.P.O. Medical Fraternity.

He was a member of the American Academy of Ophthalmology and Otolaryngology and a Fellow of the American College of Surgeons.

One daughter survives him.

Alexander Louis Prince, M.D.

Dr. Prince was born in Paris, France, on May 25, 1884, and died in Hartford, Connecticut, on May 25, 1938.

After obtaining his medical degree from the Yale Medical School in New Haven, 1910, he began the practice of medicine in New Haven in 1911. Later he took post-graduate work at the Pos-Graduate Hospital, New York, moved to Hartford, Conn., and specialized in diseases of the eye.

Dr. Prince was at one time assistant professor of physiology at the Yale University School of Medicine, was chief ophthalmic surgeon at the Hartford Hospital, and on the staffs of the Manchester Memorial Hospital, and the Windham Community Memorial Hospital at Willimantic, at the time of his death.

He was a member of the American Academy of Ophthalmology and Otolaryngology, American Collge of Surgeons, and local

and state medical societies. He was the author of many articles on his specialty.

He served during the World War as physician to Hospital No. 32 bis Passy par Veron, France, for the Fitzgerald Foundation from July to September, 1915, and when the United States entered the war he served with Mobile Hospital No. 39, Yale Unit, being discharged with the rank of Captain in January, 1919.

Dr. Prince is survived by his widow, stepmother, two half-brothers and one half-sister.

Willard Curtis Proud, M.D.

Dr. Proud was born in Atchison County, Missouri, on June 28, 1873, and died in St. Joseph, Missouri, on April 29, 1938.

He was a graduate of Kansas City Medical College and the Rush Medical College, Chicago. Later he took post-graduate work at Tulane University, New Orleans, the Manhattan Eye, Ear and Throat Hospital, and in London and Vienna clinics. In 1896 he began the practice of medicine in Oregon, Missouri, later moving to St. Joseph.

At the time of his death he was on the staff of the Missouri Methodist Hospital.

Dr. Proud was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons and member of local and state medical societies.

He is survived by his wife, two daughters and one son.

J. William Scales, M.D.

Dr. Scales was born on February 29, 1864, near Humboldt, Tenn., and died on December 4, 1937, in Pine Bluff, Arkansas.

After receiving his medical degree from Vanderbilt University School of Medicine, Nashville, Tenn., in 1888, Dr. Scales moved to Pine Bluffs, where he practiced continuously for 50 years, except during the United States' participation in the World War, when he was a Captain in the Army Medical Corps, stationed at Fort Thomas, Kentucky. For a number of years he was eye, ear, nose and throat specialist for the Cotton Belt Railway in the Pine Bluff district.

He was a member of the American Academy of Ophthalmology and Otolaryngology, and Fellow of the American College of Surgeons.

Dr. Scales was married three times, all of his wives having predeceased him, and two daughters survive.

George E. de Schweinitz, M.D.

Dr. de Schweinitz was born in Philadelphia on October 26, 1858, and died there on August 22, 1938.

He received his A.B. and A.M. degrees from Moravian College in 1876 and his Medical Degree from the University of Pennsylvania in 1881. In 1891 he became lecturer on Ophthalmoscopy in the University of Pennsylvania and Professor of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine. In succession he was Professor of Ophthalmology at Jefferson Medical College, the University of Pennsylvania, and the Graduate School of Medicine of the University of Pennsylvania, consulting ophthalmologist to several Philadelphia hospitals, and in 1929 Professor Emeritus of Ophthalmology in the Graduate School of Medicine.

He was a life member of the American Academy of Ophthalmology and Otolaryngology, Secretary of the Section on Ophthalmology of the American Medical Association in 1891-92, Chairman in 1896-97, and President of the American Medical Association in 1922-23. He served as President of the College of Physicians of Philadelphia in 1910-13, and of the American Ophthalmological Society in 1916. He was an honorary member of the Ophthalmological Society of the United Kingdom, a member of the French, British and Belgian Ophthalmologic Societies and of all International Congresses.

During the World War he was a member of the Council of National Defense in 1917, commissioned a Major on April 9, 1917, and a Lieutenant Colonel in May, 1918. Later he was officer in charge of ophthalmology and established the School of Ophthalmology in the Medical Officers' Training Camp at Fort Oglethorpe. At the end of the war he was appointed consultant in ophthalmology and a member of the editorial board of the Medical History of the War. In April, 1919, he was commissioned Colonel and in 1922 Brigadier General in the Medical Reserve Corps.

Dr. de Schweinitz was widely known for his contributions to ophthalmology, was elected a Trustee of the University of Pennsylvania in 1924 and appointed Chairman of its Board of Medical Affairs in 1928. He was a bachelor and is survived by one sister.

Few men have received and held greater honor and esteem in their own cities. Dr. de Schweinitz was the soul of honor and ethics in medical practice; his ophthalmologic contemporaries all trusted him and his juniors reverenced him. When Dr. de Schweinitz presided at dinner or banquet, the occasion was a rare treat to his associates, he presided with such courtesy, grace and tact and marvelous diction. He never pushed himself forward. When visiting a distant group of medical men or alumni, he was wont to retire in the background or corner, and the others flocked to and around him. In his later years when he shared less in the discussions, it was nevertheless part of his code to religiously attend all meetings of the Section of Ophthalmology of the College. His presence will long be missed.

Walter Karl Seelye, M.D.

Dr. Seelye was born in Eliota, Minnesota, on February 18, 1868, and died in Seattle, Washington, on June 24, 1938.

He received his medical degree from the University of Iowa College of Medicine, Iowa City in 1892, and began the practice of medicine in Streator, Illinois, in 1895. He moved to Seattle, Washington, in 1906, where he specialized in diseases of the eye, ear, nose and throat.

He was a member of the American Academy of Ophthalmology and Otolaryngology, member and one time President of the Puget Sound Academy of Ophthalmology and Otolaryngology, President of the North End Medical Society, and member of other local and state medical societies.

In addition to his specialty he was interested in the study of malingering and was for many years a consultant for the State Department of Labor and Industries.

He is survived by his former wife, one daughter, and one son, Dr. Walter Bale Seelye.

James Miller Smith, M.D.

Dr. Smith was born on September 28, 1875, and died in Jacksonville, Florida, on January 8, 1938.

He obtained his early education in Hawkinsville, Georgia, and graduated from Emory University, Atlanta, with his medical degree. Following this he took post-graduate work at the Manhattan Eye, Ear and Throat Hospital, New York City, and began to practice his specialty in Valdosta, Georgia, in 1898.

Dr. Smith was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, past President and member of the Medical Association of Georgia.

Outside of his medical work, he had many civic interests, and at the time of his death was Chairman of Lowndes County Board of Health, and Chairman of the Board of Directors of the First National Bank, Valdosta.

He is survived by one daughter.

James Alfred Spalding, M.D.

Dr. Spalding of Portland, Maine died on February 27, 1938, at the age of 91.

He was a graduate of Harvard University Medical School, Boston, in 1870, and was on the staff of the Maine General Hospital.

He was a life member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, member and past President of the Maine County Medical Society, past President of the Cumberland County Medical Society, and member of the American Ophthalmological Society.

He was well known as the author of *Life of Dr. Lyman Spalding* and *Maine Physicians of 1820*, and was editor of the Maine Medical Journal from May, 1933, to September, 1935.

George Leslie Strader, M.D.

Dr. Strader was born in Lafayette County, Missouri, on December 27, 1870, and died in Claremont, California, on January 21, 1938.

He received the A.B. degree from Macomb Normal School, Macomb, Illinois and his medical degree from the University of Nebraska College of Medicine, Omaha, in 1899.

For five years he was associated with Dr. Harold Gifford in Omaha, before moving to Cheyenne, Wyoming, in 1903 to practice his specialty.

Dr. Strader was a Life Member of the American Academy of Ophthalmology and Otolaryngology and in 1908 was a member of the House of Delegates of the American Medical Association.

He was interested in all civic and local welfare organizations, a member of the Wyoming State Legislature in 1911 and a 32nd Degree Mason, Knights Templar.

His wife and one son survive him.

Alfred Lubbert Van Dellen, M.D.

Dr. Van Dellen was born in Holland on April 25, 1887, and died at the Mayo Clinic, Rochester, Minn., on April 1, 1938.

After obtaining his medical degree from Northwestern University School of Medicine, he took post-graduate work at the Rush Medical School, Chicago, and in Vienna, Austria, and began the practice of medicine in Chicago in 1912. At the time of his death he was Clinical Associate in Ophthalmology at Rush Medical School and was on the staffs of the Illinois Eye, Ear, Nose and Throat Infirmary and the Englewood Hospital.

Dr. Van Dellen was a member of the American Academy of Ophthalmology and Otolaryngology, and local and state medical societies.

He is survived by his wife, one daughter and two sons.

John Martin Wheeler, M.D.

Dr. Wheeler was born in Burlington, Vermont, on November 10, 1879, and died at his summer home in Underhill, Vermont, on August 22, 1938.

He graduated as B.A. from the University of Vermont in 1902, received his medical degree in 1905, and degree of Master of Science a year later. He began the practice of medicine in New York in 1909.

In 1928 Dr. Wheeler was appointed Professor of Ophthalmology at Columbia University and in 1931 Director of the Institute of Ophthalmology of the Columbia-Presbyterian Medical Center. At the time of his death he was Consulting Ophthalmologist to the Presbyterian, Fifth Avenue and Bellevue Hospitals, Neurological Institute, Sloane Hospital for Women and the New York Eye and Ear Infirmary, New York City; St. Luke's Hospital, Newburgh, N. Y., and the Hackensack (N. J.) Hospital.

Dr. Wheeler was a member and past President of the American Academy of Ophthalmology and Otolaryngology, past President of the Society of Plastic and Reconstructive Surgery, member of the American Ophthalmological Society and Fellow of the American College of Surgeons.

He served during the World War with the Army Medical Corps as a Captain and later was promoted to the rank of Major.

In 1936 he was presented with the Leslie Dana Gold Medal for "outstanding achievements in the prevention of blindness and the conservation of vision" by the National Society for the Prevention of Blindness (of which he was formerly a member of the Board of Directors) in cooperation with the St. Louis Society for the Blind. He was a member of the Board of Trustees of the University of Vermont and was awarded the honorary degree of Doctor of Science by his Alma Mater in 1928, and by Middlebury College (Vermont) in 1933.

When Dr. Wheeler relinquished private practice to take up the important work of the direction of the Institute of Ophthalmology at the Medical Center, he is said to have sadly missed the familiar contacts with his patients who had become personal friends. His unobtrusive manner, twinkling eyes and whimsical humor were a magnet ever increasing his horde of friendships. A most touching tribute is the letter from the National Society for the Prevention of Blindness, enclosing a copy of his personal note written late in July, appealing to the world for support of this great work for the prevention of blindness. His influence will carry on.

Dr. Wheeler accepted the responsibility of many offices without a murmur, felt most deeply the honor of the Presidency of the Academy and enjoyed its association.

While the public perhaps measured his fame by his widely known operative work, we of the Academy loved and honored John Wheeler for himself alone.

Charles Peter White, M.D.

Dr. White of Wilmington, Delaware, died on April 13, 1938, at the age of 67.

He received his medical degree from the University of Pennsylvania Department of Medicine, Philadelphia 1902, and at the time of his death was on the staffs of the Delaware and Wilmington General Hospitals, and ophthalmologist to the Delaware State Hospital, Farnhurst.

He was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, past President of the Medical Society of Delaware and past President and Secretary of the Newcastle County Medical Society.

John Robert Wilson, M.D.

Dr. Wilson was born in Bennington, Vermont, on February 22, 1880, and died there on June 28, 1938.

After receiving his medical degree from the College of Physicians and Surgeons, Columbia University, in 1905, he took postgraduate work in eye, ear, nose and throat at the New York Post-Graduate Hospital, at the University of Vienna and Vienna Polyclinic, and began the practice of his specialties in Bennington, Vermont. He was in charge of the Eye, Ear, Nose and Throat Department at the Henry W. Putnam Memorial Hospital, Bennington.

He was a member of the American Academy of Ophthalmology and Otolaryngology.

He is survived by his wife.

Harry Vanderbilt Wurdemann, M.D.

Dr. Wurdemann died in Seattle, Washington, on January 30, 1938, at the age of 72.

He was a graduate of Columbian University Medical Department, Washington, D. C., in 1888, practiced in Milwaukee from 1890 to 1909 and in Seattle since 1909.

He was professor of ophthalmology at the Chicago Eye, Ear, Nose and Throat College from 1894 to 1909, and at the Milwaukee Medical College from 1900 to 1909.

Dr. Wurdemann was a member of the American Academy of Ophthalmology and Otolaryngology, Fellow of the American College of Surgeons, past President of the Puget Sound Academy of Ophthalmology, member of the Pacific Coast Oto-Ophthalmological Society, advisory member of the National Committee for Prevention of Blindness.

He served as Medical Examiner in Aeronautics for the U. S. Department of Commerce and as flight surgeon in the U. S. Army Air Corps, and was formerly President of the Reserve Officers' Association of Washington.

Dr. Wurdemann contributed frequently to scientific periodicals, was the author of chapters in textbooks on Ophthalmology, was at various times editor of the Annals of Ophthalmology, managing editor of Ophthalmology, associate editor of the Ophthalmic Record and the American Journal of Ophthalmology. He served as Secretary of the Section on Ophthalmology of the American Medical Association in 1894-1895, as Chairman in 1899-1900, and as member of the House of Delegates in 1902, 1906 and 1914.

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